TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE

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Submitted By

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Dedicated

to

my Parents

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-Dr Jadhao Atul Rakhmaji

LIST OF ABBREVIATIONS

CSF	Cerebrospinal fluid
CT	Computed Tomography
DC	Decompressive Craniectomy
ED	Emergency Department
EVD	External Ventricular Drain
GCS	Glassgow Coma Scale
GOS	Glassgow Outcome Scale
HTN	Hypertension
ICH	Intracerebral hemorrhage
ICP	Intracranial pressure
ICU	Intensive care unit
IVH	Intraventricular hemorrhage
mRS	Modified Rankin scale

ABSTRACT

Introduction: Intracerebral Haemorrhage is a deadly illness that has a high death rate and morbidity. Intracerebral haemorrhage (ICH) ranks second among pathologies underlying stroke. The Incidence is 10-40 per year per lakh population. Arterial hypertension is reported as the main cause of ICH – 57% in meta-analysis. The outcome of Hypertensive ICH is determined mainly by severity of bleeding. Age, level of consciousness, volume of hematoma and presence of intraventricular blood are independent predictors of recovery. The impact of surgical evacuation of hematoma on the prognosis, selection criteria and timing of evacuation has to be assessed.

Aims & objectives: To Study the Outcomes of Surgical Interventions in Patients with Hypertensive Supratentorial Intracerebral Haemorrhage.

Material & methods: The study was conducted at Dayanand Medical College and Hospital from 1st December 2022 to 30th November 2023. This follow-up study included 50 patients of either sex with diagnosis of hypertensive supratentorial ICH and operated for the same by different surgical interventions. Patients were operated and GCS, GOS, mRS at discharge and at 2 months were done to analyze the clinical outcome. The data was collected, analyzed All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21 version (SPSS Inc., Chicago IL, USA) statistical program for Microsoft windows.

Results: The average age was 53.58 years (median 52 years) with SD of 12.92 years. This study found male preponderance, with a male to female ratio of 5.25:1. Hypertension was present in majority of the patients 38 (76%), Diabetes Mellitus in 8 (16%), coronary heart disease in 3 (6%), Chronic Kidney Disease and Hypothyroidism was present in 4(8%) patients. In this study there was statistically strong correlation between GCS at admission, at discharge and on follow up at 2 months. There is statistically significant relationship between IVH and GCS at discharge. In our study Mid-line shift has statistically strong correlation with clinical outcome (GOS, mRS) and mortality. Operative techniques used in our study have strong correlation with clinical outcome and mortality

Conclusion: In conclusion, an elderly male with associated comorbidities mainly Hypertension, Diabetes mellitus and Chronic alcoholic patients are at more risk to develop ICH. Clinical presentations including low GCS, Neurological deficit, Headache and Loss of consciousness have poor clinical outcome. The most common location for ICH is Gangliocapsular region followed by Thalamic region; however, the specific location of the hematoma doesn't influence overall clinical outcome or mortality rate. Maximum patients have hematoma volume less than 30 ml in the study and ICH volume plays a key role in clinical outcome of the patient. Midline shift has impact on clinical outcome and mortality i.e. patients with more midline shift has poor clinical outcome. Various operative techniques employed in managing ICH have shown significant effects on clinical outcome and mortality.

Keywords: Intracerebral haemorrhage(ich), Supratentorial, GCS, Gos, mRS

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INTRODUCTION

Intracerebral Haemorrhage is a deadly illness that has a high death rate and morbidity. Intracerebral haemorrhage (ICH) ranks second among pathologies underlying stroke.[1] The Incidence is 10-40 per year per lakh population.[2]

Spontaneous ICH constitutes about 10-20 percent of the total number of strokes. A considerable number of patients with ICH is misdiagnosed as brain infracts [3] resulting in an underestimation of the incidence of ICH in any community. Now more than half of the acute stroke patients are investigated within 3 days resulting in identification of more haemorrhagic strokes.

Within a year, 58% of ICH patients pass away, and 2/3 of those who survive end up moderately or severely impaired.[4] There is a notable variation in the incidence of ICH between nations and ethnic groups. Primary ICH incidence rates were twice as high in low- and middle-income nations as they were in high-income nations.[5] Black Americans had a greater incidence of ICH than White Americans did in a population-based US investigation that identified 1,038 hospitalized patients for ICH (48.9 vs. 26.6 per 100,000 person-years). The incidence in Asian communities is almost twice that of white Western populations.[6] As people age, the incidence of ICH rises. The onset occurs most often during activity (rarely during sleep) and is linked to either an increase in cerebral blood flow or blood pressure.

Modifiable risk factors include high BP, tobacco smoking, consumption of alcohol, decreased LDL (Low-density) lipoprotein cholesterol, low triglycerides and drugs including anticoagulant, antithrombotic agent and sympathomimetic.

Non-modifiable risk factors include male sex, old age, Cerebral Amyloid Angiopathy and Asian ethnicity.

Arterial hypertension is reported as the main cause of ICH – 57% in meta-analysis.[7] The frequency varies considerably. Neurosurgical case series show more hematomas from aneurysms and arteriovenous malformations (AVM) than from hypertension.[8] More anticoagulant

bleeds will be observed where anticoagulant therapy is widely used. Charcot and Bouchard (1868) [9] described miliary aneurysms, which is the pathological substrate of many of these haemorrhages. The underlying pathology is lipohyalinosis and fibrinoid necrosis.[10]

The outcome of this common and distinct entity of Hypertensive ICH is determined mainly by severity of bleeding.[11] Previous multivariate studies principally investigated short term outcome 30-day mortality [12] after ICH and showed that age, level of consciousness, volume of hematoma and presence of intraventricular blood are independent predictors of recovery.[13]

It's unclear whether alcohol consumption, smoking, use of NSAIDs or clinical parameters like level of blood pressure, target organ damages like hypertensive changes in optic fundus and left ventricular hypertrophy would influence the outcome.

These factors might also impair the outcome by making patients prone to severe initial bleeding, to enlargement by hematoma or to cerebral ischemia. Further, prognostic significance of signs of tentorial herniation, midline shift and interference of the hematoma with the CSF circulation has to be evaluated. The impact of surgical evacuation of hematoma on the prognosis, selection criteria and timing of evacuation has to be assessed.[14]

Common sites for ICH

- Striate body (Basal ganglia): Putamen most common; also include: Lenticular nucleus, Internal capsule and Globus pallidus - 50%
- Thalamus - 15%
- Pons (90% of these are genuinely hypertensive) - 10-15%
- Cerebellum - 10%
- Cerebral white mater - 10-20%
- Brain stem - 1-6%

Management

Conventional medical therapy of haemorrhagic stroke is principally focused on adjunctive measures to minimize injury and to stabilize individuals. General measures to maintain airway and hydration, control blood pressure, reduce ICP, prevent seizures and maintain systemic health are important in preventing progression of the hemorrhage, edema and brain ischemia. [15,17]

The goals of surgical evacuation of hematoma are:

- To reduce the mass effect
- Block the release of neuropathic products from the hematoma
- Prevent prolonged interaction between the hematoma and the normal tissue which can initiate pathologic process.

Some patients have a penumbra of potentially viable but functionally impaired tissue around the ICH. Surgical evacuation of clot may improve the function and recovery in this penumbra.

Surgical interventions in SICH include:

- Craniotomy and hematoma evacuation.
- Decompressive craniectomy and hematoma evacuation. [18,19,20]
- Minimally invasive techniques Such as endoscopic hematoma evacuation and stereotactic hematoma evacuation. [21]
- Burr hole and EVD of acute hydrocephalus, fibrinolysis of IVH using ‘Recombinant tissue plasminogen activator’ (rtPA), or streptokinase are other indications for surgery, especially in IVH

The largest trial so far has been “The International surgical trial” in spontaneous ICH which has been published in 2005. A recent meta-analysis collecting the data from 8 RCT’s since 1985 suggests improved surgical outcome if performed in less than 8 hours of ictus, in hematomas between 20-50ml in GCS 9-12 and in patients between 50-69 years. In excess of this, there was evidence that more superficial hematomas with no IVH might also benefit.

The goal of "The International Surgical Trial in Intracerebral Haemorrhage" (STICH) was to determine whether conservative medical care or early hematoma evacuation (in less than 24 hours of

randomization) was superior. A total of 1,033 patients were randomly assigned to either the early surgery (n = 503) or initial conservative treatment (n = 530) groups after being recruited from 83 centres across 27 nations. Although there was no overall statistically significant difference in mortality or functional result between the early surgery and initial conservative therapy groups, the research did suggest that early surgery may be helpful in some patients with superficial lobar hemorrhage.

The results of "STICH II" trial' was published in 2013.[22] In conscious patients with superficial lobar hemorrhage of 10-100 mL within 1 cm of the cortical surface, without IVH, and admitted within 48 hours after the onset of symptoms, this experiment evaluated the results of early surgery vs initial conservative care. A total of 601 patients were randomly assigned to the early surgery (n = 307) or initial conservative therapy (n = 294) groups after being enrolled from 78 centres across 27 nations. In the early surgery group, the 6-month mortality rate was 18%, while in the initial conservative care group, it was 24% (OR, 0.71; 95% CI, 0.48-1.06; P=0.095).

The results of “The STICH II trial” show that early surgery doesn’t raise the risk of death or impairment at six months, and that patients who have spontaneous superficial intracerebral hemorrhage without intraventricular hemorrhage may have a slight but meaningful survival advantage. Hematoma evacuation may be deemed a life-saving intervention for individuals exhibiting neurological decline due to supratentorial ICH.

A number of studies indicate that MIS (minimally invasive surgical evacuation) may be less invasive and yield better results than craniotomy. [23]A recent study, "The Minimally Invasive Surgery Plus Recombinant Tissue-Type Plasminogen Activator for ICH Evacuation Trial II" (MISTIE II), found that the hematoma evacuation group experienced a statistically significant decrease in perihematomal edema.

AIMS AND OBJECTIVES

“To Study the Outcomes of Surgical Interventions in Patients with Hypertensive Supratentorial Intracerebral Haemorrhage.”

REVIEW OF LITERATURE

1. Epidemiology:

Definition

Intracerebral haemorrhage (ICH) literally means haemorrhage in brain. ICH is defined as “extravasation of blood into brain parenchyma”.[24] It results from rupture of intracerebral artery, when bleeding occurs into brain parenchyma leads to hematoma formation. Haemorrhage can also occur into the ventricles, causing intraventricular haemorrhage (IVH).

Incidence

ICH accounts for 10-30 % in all types of strokes. [25-27] In contrast to past advances with ischemic stroke prevention, ICH incidence has remained steady.[28] According to ethnic origin, a fairly recent meta-analysis of 36 studies from 20 countries reported the incidence of ICH is 24.2 cases per 1 lakh population per year in white people, 22.9 in black people, 19.6 in Hispanic people, and 51.8 in Asian people.[28] These changes partly can be explained by differences in educational attainment, socioeconomic status, and prevalence of vascular risk factors, such as hypertension, smoking, and alcohol consumption.[29,30,31] Overall ICH incidence is 24.6 (range from 1.8 to 129.6) cases per 1 lakh population per year, a discovery by the same meta-analysis, having remained steady since 1980, and having decreased only in few populations with improved access to medical care and blood-pressure control.[28,32,33]

Incidence is higher among males and increases with age: from annual 1.9 cases per 1 lakh among people aged under 45 to 196.0 cases among those over 84.[28] Studies have shown 44.4 % of all ICH occurring at age over 60, median age of ICH patients being 61, and incidence rates doubling every 10 years after age 35. [34,35] The 30-day mortality ranges from 32-35%. Of the total patients with ICH only 20% of patients are expected to be functionally independent at the end of 6 months.

Classification of Spontaneous ICH:

Classification of spontaneous ICH is confusing, as often times the knowledge on the underlying pathology and exact source of bleeding is unknown [36]

Johann Jakob Wepfer based in Germany and Switzerland in the 17th century was the first to make note that stroke can be caused by either hemorrhage or clotting [37]. In the next century, Italian Morgagni of Padua, worked on post-mortem studies and linked them with different clinical stroke subtypes [37,38].

Huge progress has been made in the diagnosis and management of stroke in the past few decades. Just almost fifty years ago, the standard procedure to differentiate brain hemorrhage from ischemic stroke was a lumbar puncture to detect blood in the cerebrospinal fluid. In later years, midline echogram (transcranial sonography i.e. ultrasound-based imaging technique) could be used to detect displacement of the midline structures due to unilateral lesion in the brain. Afterwards, the diagnostics of ICH were revolutionized by advanced imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), which in addition to showing the hematoma itself, may help in deciphering the etiology of the bleeding.[37,39,40]

Afterwards, the diagnostics of ICH were revolutionized by advanced imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), which in addition to showing the hematoma itself, may help in deciphering the cause of the bleeding. Spontaneous ICH can be classified as primary or secondary depending on the underlying cause. Primary accounts for approximately 70-80% of cases and is due to spontaneous rupture of small vessels damaged by HTN or amyloid angiopathy (AA).

Primary Intracerebral Hemorrhage-

This is the mundane but often devastating "spontaneous" brain hemorrhage. It originates from a rupture of small cerebral penetrating arteries and predominantly a result of chronic HTN and degenerative

changes in these arteries.[41] Primary ICH is classified according to location as lobar or non-lobar and supratentorial or infratentorial.[42]

In order of frequency, most common sites of a cerebral hemorrhage are:

1. Putamen and adjacent internal capsule (50%)
2. Central white matter of the temporal, parietal or frontal lobes (10-20%) (Lobar hemorrhage, not strictly associated with hypertension)
3. Thalamus (15%%)
4. Cerebellar hemisphere (10%) and
5. Pons (10-15%).

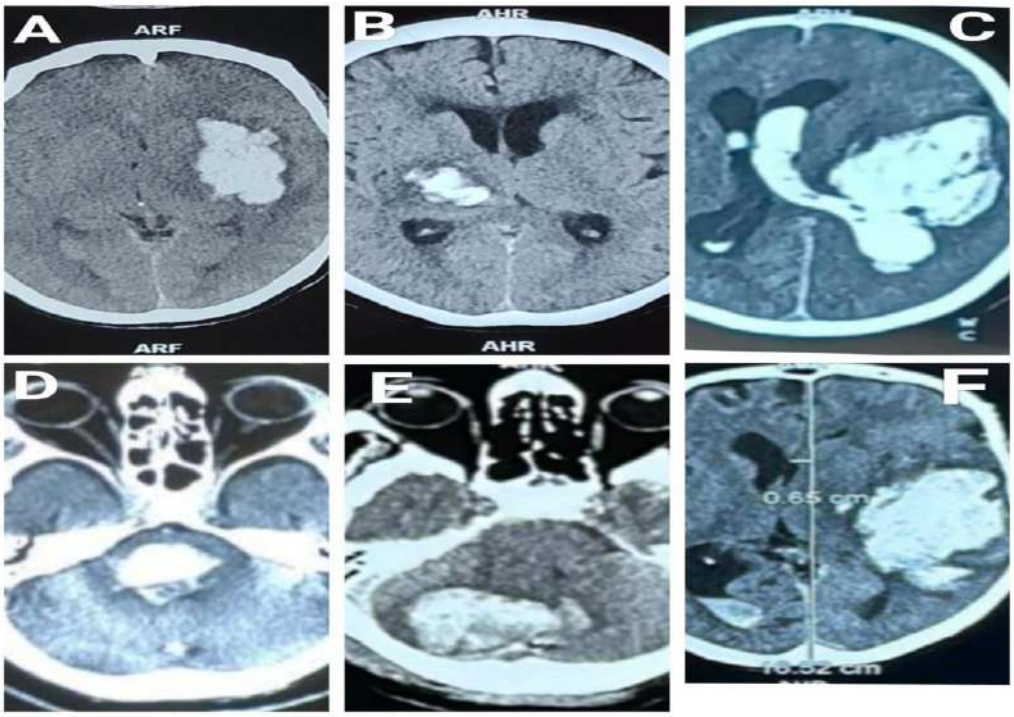


Photo 1: Common locations of Spontaneous ICH are Putamen(A), Thalamus(B), Subcortical white mater(C), Pons(D), Cerebellum(E), Lobar ICH(F)

Approximately 2 percent of primary hemorrhages are multiple. Multiple simultaneous intracerebral hemorrhages may raise the possibility of amyloid angiopathy or bleeding diathesis but may occur when one conventional hypertensive intracerebral hemorrhage causes hypertension, which in turn results in one or more additional hemorrhages.

There are two types of vessel pathologies accounting for most primary intracerebral hemorrhages:

- Deep perforator arteriopathy (hypertensive arteriopathy) >50% [43] and
- Cerebral amyloid angiopathy (CAA) 30% [44]

Hypertensive arteriolopathy is characterized by loss of smooth muscle cells from the tunica media, deposits of fibro -hyaline material, narrowing of the lumen and thickening of the vessel wall; additionally, microatheromas and microaneurysms are possible. [45] CAA is caused by beta amyloid deposits within cortical and leptomeningeal arteries.[46]

Secondary Intracerebral Hemorrhage:

Secondary ICH is associated with a number of congenital and acquired conditions such as vascular malformations, neoplasms, infarct with haemorrhagic transformation, coagulation disorders, anticoagulants, thrombolytic agents, cerebral vasculitis, drug abuse and cerebral venous thrombosis [47]. The prevalence of AVM (abnormal connection between arterial and venous systems without the normal capillary bed) is approximately 0.2% in adults and the annual risk of hemorrhage in unruptured AVM is approximately 2%. The prevalence of cavernomas (cluster of thin- walled vessels without elastic fibers or smooth muscle, commonly surrounded by a rim of hemosiderin-laden gliotic tissue) is slightly higher than AVM, approximated 0.3-0.6%, but the risk of first bleeding is lower, around 0.4-0.6% per year.

2. Etiology:

Hypertensive vasculopathy is most common etiology of spontaneous ICH. Cerebral amyloid angiopathy and vascular malformations are the most common cause of non-traumatic lobar ICH in elderly and children respectively.

Hypertensive ICH occurs in the territory of penetrating arteries often at 90-degree angle with the parent vessel. These small penetrating arteries may be particularly susceptible to the effects of hypertension because of pressure of the much larger parent vessel directly [48]. The

blood vessels with hypertensive hemorrhage usually are similar to hypertensive occlusive disease and diabetic vasculopathy, which cause lacunar strokes. These vessels supply the pons and midbrain (penetrators of the basilar artery), Thalamus (thalamostriate penetrators off the P1 and P2 segments of the posterior cerebral arteries) and putamen and caudate (Lenticulostriate penetrators off the M1 segment of the MCA-Middle cerebral artery).

Hypertensive vasculopathy is believed to play an important role in the development of white matter disease, which may explain the finding of correlation between white matter disease and risk of ICH.[49] The combination of Pathologic examination of the blood vessels in chronic hypertension and ICH disorder creates hypertensive hemorrhage theory. The development of penetrator vessels in intimal hyperplasia with hyalinosis can create focal necrosis and broken in the wall of the vessel. These microscopic "pseudo-aneurysms" with subclinical leaks of blood may be relatively common. When the clotting system is unable to control vessel wall problem create massive hemorrhage.

Cerebral amyloid angiopathy - Cerebral amyloid angiopathy (CAA), although usually asymptomatic, is an important cause of primary lobar ICH in elderly people. CAA causes weakness in the structure of the vessel walls and bleeding because of the deposition of congophilic material in small to medium-sized blood vessels of the brain and leptomeninges. Manifestation of CAA is spontaneous lobar hemorrhage. The common location of CAA-related ICH is in the putamen, thalamus and pons.

Other causes of non-traumatic ICH include [50]:

- Haemorrhagic infarction (including cerebral venous thrombosis)
- Septic embolism, mycotic aneurysm
- Brain tumor
- Bleeding disorders, liver disease, thrombolytic therapy
- Central nervous system infection (e.g., herpes simplex encephalitis)
- Moyamoya disease

- Drugs (Cocaine, Amphetamines)

3. **Risk factors:** It includes
   - Hypertension
   - Older age
   - Presence of amyloid angiopathy (Cerebral)
   - Use of anticoagulant therapy.

Hypertension- Hypertension is the very important risk factor for development of ICH. Hypertension doubles the chance of development ICH. [51,52] The finding of study of Ahmadi Ahangar et al in Iran in 2018 indicates Hypertension was most frequent stroke risk factor especially in ICH and male patients. Of 61 case of ICH, 37.7% cases involved hypertension. [53] Also, in prognosis of ICH patients were influential. [54]

Anticoagulants - Anticoagulation with warfarin raises the chance of ICH 2-5 times, depending upon the intensity of anticoagulation. [55,56] Retrospective studies explains that warfarin therapy with an INR more than 3 increases the risk for larger initial haemorrhage volume and also poorer outcome after ICH.

Antiplatelet - There is small absolute increase in the chance of development of primary ICH associated with the use of aspirin or antiplatelet agents, based on meta-analyses of randomized, controlled trials, although other case-control studies have not found an increased risk.[57]

Others- Include High alcohol intake, Black ethnicity, Lower cholesterol and LDL levels and genetic variation.

High alcohol intake significantly increases the chances of development of ICH which is nearly 3 times the normal.

Black ethnicity is also associated with increase in the chance of development of ICH that is age-related. Findings from a surveillance study and a prospective cohort have found that the risk factors of race and age appear to interact, such that young (45 to 60 years) blacks have higher risk of ICH than whites, but this increased risk declines with increasing age. [58]

Smoking, in study conducted by Ahmadi Ahangar et al in Iran, indicate that high smoking was seen in ICH.[53]

Lower cholesterol and lower LDL are associated with an increased chances of development of ICH. 'A systematic review and meta-analysis' of 23 prospective studies found an inverse relationship between total and LDL-cholesterol and the risk of ICH. However, most systematic reviews and meta-analyses have found that the treatment with statins doesn't increase the risk of primary ICH or to negatively impact prognosis. [59]

4. Pathophysiology of ICH:

Instead of a single event, ICH is a dynamic process with three phases:

- Initial blood eruption
- Hematoma expansion
- Development of perihematomal edema. [60-63]

Since the majority of these hematomas are caused by the rupture of an artery or arterioles, the pressure inside the hematoma initially equals the pressure in the surrounding arterial blood. The surrounding brain parenchyma is compressed as a result of the increase in the pressure, which causes ischemia. An area of total ischemia and an area of oligemia surround hematomas; these areas are similar to the penumbra of an occlusive stroke.

Experimental evidence shows that area of ischemia is much more than the hematoma. In addition to the mechanical compression of the surrounding microcirculation, blood's vasospastic feature most likely contributes.[64] Rebleeding has been demonstrated to be linked to a poorer prognosis. Both focal (caused by ischemia) and global (caused by decreased cerebral perfusion pressure brought on by an increase in intracranial pressure) damage are possible.[65]

Perihematomal edema develops over many days and is primary cause of neurological deterioration after first day from ictus, by interfering with functioning of nearby brain areas. It causes relative ischemia by compressing blood vessels, and by increasing intracranial pressure. [66,67] It derives from plasma, is caused mainly by the inflammatory response secondary to local release of thrombin and other end

products of coagulation from the hematoma, by cytotoxic mediators, by disruption of the blood-brain-barrier, sodium pump, and neurons. [68-71]
Initial factors responsible for edema include:

- The release of Arachidonic acid and free radicle.

When a large edema that surrounds these lesions, different agents may exert their effects. Kase et al (1991) [72] added immune component to the pathogenesis of this edema.
Glutamate is one of the more excitatory amino acids which is increased. Leukotrienes and kinins also contribute to the progression of edema. Ultimately, the calcium influx will be the cause of neuronal death. Numerous medicines were tested in the treatment of this cascade of events in the marginal zone, which is most likely to provide clinical benefit to the patients. Superoxide dismutase, calcium antagonists, steroids, NMDA receptor blockers (both competitive and non-competitive), and even immunosuppressive drugs. It is still not clear how beneficial they will be in treating these people.

5. Clinical presentation:

Large ICH usually produce catastrophic acute symptoms. Onset is often associated with physical or emotional activity: onset during sleep is rare. [73,74] Common early features include alterations in consciousness, nausea and vomiting. Unconsciousness, headache, Vomiting, neck stiffness and seizure occur more with ICH than brain infarcts. TIA is more with infarct now less severe dysfunction clinically indistinguishable from ischemic stroke is also found out to be caused by ICH by CT scan and MRI.

Onset and progression

The most cases take place during routine activity. The neurologic signs and symptoms usually increase gradually over minutes or a few hours, in contrast to brain embolism and subarachnoid haemorrhage where the neurologic signs and symptoms are often maximal at onset. However, some patients with ICH are comatose when first discovered or upon arrival to the emergency department.
Headache, vomiting and decreased level of consciousness develops in large hemorrhage. Headache and vomiting occur in approximately half

of the patients with Intracerebral hemorrhage (ICH). Headache may be due to traction on meningeal pain fibers, increased intracranial pressure (ICP) or blood in cerebrospinal fluid (CSF); it is most common with cerebellar and lobar hemorrhages. These symptoms are absent with small hemorrhages; the clinical presentation in this setting is that of a gradually progressing stroke.

Patients may complain of neck stiffness and have signs of meningeal irritation on physical examination, if there is intraventricular blood. The mass effect caused by the

bleeding in parenchyma of the brain causes damage by compressing local structures and also raises the ICP. [75,76] Increase of global ICP can cause herniation and cause compression of the arteries, leading to secondary ischemia. Stupor or coma in ICH is an ominous sign. The only exception is that, patients with thalamic hemorrhage, in whom involvement of the reticular activating system is the cause of stupor rather than diffuse brain injury; these patients may be recovered after blood is reabsorbed. The signs and symptoms of hypertensive ICH vary according to the location and size of the haemorrhage.

Putaminal hemorrhage - Spread of hemorrhage into the putamen mostly occurs along white matter fiber tracts, causing hemiplegia, hemisensory loss, homonymous hemianopsia, gaze palsy, stupor and coma. Aphasia, neglect and hemianopia indicate dysfunction of the higher-level cortical structures.

Internal capsule hemorrhage - small hemorrhages restricted to the internal capsule may cause mild dysarthria, contralateral hemiparesis and sensory deficit. [77]

Cerebellar hemorrhage - Cerebellar hemorrhage usually originates in the dentate nucleus, extends into the hemisphere and fourth ventricle and possibly into the pontine tegmentum. Symptoms include inability to walk due to imbalance, vomiting, headache, neck stiffness, gaze palsy and facial weakness without hemiparesis. In cerebellar hemorrhages the local compression leads to obstruction of the aqueduct, leading to obstructive hydrocephalus and that's why cerebellar hemorrhage is a

crucial diagnosis to make since these patients frequently deteriorate and require surgery. [78]

Thalamic hemorrhage - Thalamic hemorrhage may extend in transverse direction to the posterior limb of the internal capsule, downward to put pressure on the tectum of the midbrain or may rupture into the third ventricle. Symptoms include hemiparesis, hemisensory loss and occasionally transient homonymous hemianopsia. There may also be an upward gaze palsy with miotic pupils that are unreactive, peering at the tip of the nose, skewed or "wrong way eyes" toward the weak side (in contrast to hemispheric cortical injury in which the eyes are deviated away from the hemiparesis). Aphasia may occur if the bleed affects the dominant hemisphere, while neglect may develop if the bleed affects the nondominant hemisphere. Mortality rate in Thalamic hemorrhage is also higher significantly. [79]

Lobar hemorrhage - Lobar hemorrhages vary in their neurologic signs and symptoms depending upon location. They most often affect the parietal and occipital lobes. These bleeds are associated with a higher incidence of seizures. Occipital hemorrhages frequently present with a very dense contralateral homonymous hemianopsia. Hemorrhages in the frontal region will bring about a contralateral plague or paresis of the leg with relative sparing of the arm.

Pontine hemorrhage - Pontine haemorrhage may extend into the base of the pons. These often lead to deep coma over the first few minutes following the hemorrhage, probably due to disruption of the reticular activating system. The motor examination is marked by total paralysis. The pupils are pin-point and react to a strong light source. Horizontal eye movements are absent and there may be ocular bobbing, facial palsy, deafness and dysarthria when the patient is awake.

6. Early neurological deterioration and hematoma expansion:

ICH patients are relatively more prone to experience an early decline in their neurological condition, which is typically the result of an early hematoma growth or the development of acute hydrocephalus due to IVH. [80,81] Studies vary in their definitions of early neurological

deterioration, but generally speaking, it is understood to mean either a decline from the initial neurological examination (e.g., a change in the initial scores for the GCS or NIHSS) or progression towards death. [82]

Up to 40% of patients experience an early decline in their neurological condition in less than 48 hours, and this is associated with poor long-term prognosis. A prospective cohort study involving 46 non-comatose patients (GCS > 8) with ICH was reported by Mayer et al. [80] Neurological deterioration occurred in 15 patients (33%) of whom 8/15, or 53%, experienced it on their first day of admission. On the initial CT scan, patients with neurological deterioration exhibited greater mass effects (60 % vs. 19%, $p < 0.01$) and hematoma volumes (mean volume: 45 ml vs. 16 ml, $p < 0.01$). In patients with neurological impairment, the 30-day mortality was approximately 47%, but in patients without acute worsening, it was only 3% ($p = 0.001$).

Two most important predictors on hospital admission of early neurological deterioration are:

- Hematoma volume
- Presence of IVH

Other factors are:

- Concentration of glucose
- Fibrinogen levels
- Elevated SBP [82]

The primary determinant of early neurological impairment, poor prognosis, and mortality is hematoma growth. [83,84] Brott et al. discovered that between baseline and 1-hour CT scans (i.e., within 4 hours of ictus), 26% of patients with ICH experienced significant hemorrhage expansion, which was around 233% rise from baseline hematoma volume. Furthermore, between the 1-hour and 20-hour CT scans, hematoma growth occurred in 12% of the patients.

Early neurological decline was frequently related with hemorrhage expansion. Regardless of how hematoma enlargement is defined, it is an independent predictor of death and worse outcomes. Early hematoma formation and the ensuing decrease in brain activity highlight the importance of routine neurological examination and early

repeat CT scans, which may alter the course of a patient's medical and surgical care. [85]

Predictors of hematoma expansion:

Finding the individuals who are most at risk of expansion of hematoma may be helpful since it is a significant role in determining mortality and functional prognosis. Many scores have been studied to predict hematoma expansion in ICH. Prediction scores share several common factors:

- Shorter time from ICH onset to CT
- Warfarin use
- Evidence of spot sign on CTA.

The risk of expansion of hematoma varies from 3.4-7.1 % in patients with no risk factors to 70-85.5 % in patients obtaining the maximum score. [86-88]

1. **Time from ICH onset to CT: -** Early in the course of ICH, hematoma expansion occurs. Between baseline and 1-hour CT scans, 26% of substantial hematoma growth occurs, compared to 12% between 1-hour and 20-hour CT scans. Given that CT scans conducted more than 6 hours after the commencement of ICH would likely miss any pre-existing hematoma growth,[89] it is not surprising that a shorter time interval between the ICH and CT scan would appear to be a predictor of hematoma expansion.
2. **Patients on warfarin: -** (or with INR > 1.5) have an adjusted or > 4.04 (95 % CI 1.85-9.13) have more chances of hematoma expansion as compared with patients not on warfarin
3. **Spot sign on CT Angiogram (CTA): -** It is the hematoma's focal areas of enhancement on CTA. A number of technical aspects of imaging acquisition, including the concentration of the contrast agent, the speed of the scanners, and crucially, the timing between the contrast injection and the image acquisition, affect the spot sign's identification and sensitivity in predicting hematoma expansion. [90,91]

Clinical implications of spot sign on CT Angiogram:

- The spot sign and the detection of contrast extravasation are reliable, independent indicators of hematoma growth. Nevertheless, the presence of the Spot sign doesn't always signify a definitive hematoma growth. A score for spot signs has been created to forecast the expansion of hematomas. The greatest axial dimension (1-4 mm or ≥5 mm), the number of spot signs (1-2 or ≥3), and the maximum attenuation in Hounsfield Units (120-179 HU or ≥180 HU) are all included in the score. A score of 0 denotes the absence of any spot sign on the CTA and is related to the lowest possible risk of hematoma growth (2%). The chance of expansion of hematoma is about 100% in patients who receive the highest possible score of four points.
- CTA spot sign is seen to be related to the functional outcome and mortality.
- Patients with positive spot signs on CTA may be beneficial from more intensive care to stop the expanding hematoma, which could lower mortality and enhance the patient's functional result.

7. Management of Intracerebral haemorrhage

Acute treatment of ICH in intensive care stroke unit (ISCU) instead of general intensive care unit (ICU) or general ward has been consistently proven to reduce death and dependency, and is therefore strongly recommended. [92-95] Reasons being the unified treatment strategies, dedicated multiple teams having exposure with the interactions between the injured brain and internal organ systems, and early access to stroke rehabilitation services. [96,97] Various resources needed to treat patients with ICH include Neurology, Neurosurgery, Neuroradiology, and Critical care facilities. Medical complications, such as infections and venous thrombotic events are largely preventable, and often serve as a measure of quality of standards in institutions with a stroke center designation. Combined treatment in ICSU and short-term ward with supported early discharge has been proposed to reduce mortality and complications in comparison to general medical ward. [98]

Prehospital Management

ICH is a medical emergency and therefore any delays in treatment may result in worse outcomes. Initial prehospital treatment must be focused on respiratory and cardiac management by maintaining airways, breathing, and circulation to prevent decreased oxygen supply to the brain and limit hematoma growth while maintaining cerebral perfusion. Quick delivery to the nearest emergency department (ED) is imperative in patients with abrupt neurological focal deficit, such as hemiparesis, impaired consciousness, or severe headache.[99]

Initial Management in The Emergency Department

It is of utmost importance that ED is prepared to treat patients with ICH or has a plan for transfer to a tertiary center. Majority of patients are unstable in the early phase and require close monitoring, particularly on cardiovascular, respiratory, and neurologic status. Respiratory and hemodynamic may be compromised in patients with impaired consciousness. Cardiovascular instability is responsible for increase in ICP which needs immediate attention to avoid the damage caused by hypertension or hypotension in a patient with limited autoregulation.[100] Early treatment should include stabilization of cardiorespiratory function and treatment of intracranial complications.

Intubation is needed, if other measures are insufficient to protect airways and sustain adequate ventilation with decreasing consciousness (GCS≤8) or decreased reflexes protecting the airways. Approximately 30% individuals with supratentorial ICH and majority of the pts infratentorial ICH require intubation. [101]

Urgent brain CT scanning upon arrival to the ED is vital to differentiate between ischemic stroke, subarachnoid hemorrhage, and intracerebral hemorrhage, and to evaluate the characteristics of bleeding, such as location and volume and also to detect presence of IVH, edema, increased ICP, obstructive hydrocephalus, and imminent brain herniation.

Reversal of Anticoagulation

Anticoagulant use has increased in the last few decades which caused 3-fold increase in the occurrence of anticoagulant-related intracranial

haemorrhage.[102] Patients having ICH on antithrombotic agents had raised chances for expansion of hematoma and increased chances of death and poor outcome. [103,104]

In anticoagulant associated ICH, the main goals are:

- Withholding the culprit drug
- Reversing the drug effect by the administration of antidote when available
- Monitoring the effectiveness of anticoagulation reversal with laboratory tests.

Vitamin K promotes the synthesis of endogenous clotting factors, which has a long-lasting impact. However, because it takes six hours to take action, vitamin K antagonist reversal should also be accomplished immediately with fresh frozen plasma (FFP) or prothrombin complex concentrate (PCC). Factors II, VII, IX, and X are present in FFP. Due to its brief duration of action, high amounts can be needed. There is a risk of heart failure or elevated intracranial pressure when a patient has low cardiac reserve or intracranial compliance, which makes it impossible to administer a sufficient volume of FFP quickly enough. High amounts of factors II, IX, and X, with or without factor VII, are found in PCCs, which are plasma products. [107-110] Compared to FFP, PCC can correct INR more quickly and effectively stop the formation of hematoma and also improve patient outcome. [111]

Although recombinant factor VIIa is a novel option for quickly reversing vitamin K antagonist anticoagulation, more RCT's are required to validate its application in this context. [112-116] In patients who are at high chances of developing thrombotic events, such as those who have prosthetic valves, anticoagulation can be resumed 7 to 14 days following the ICH, per the guidelines of 'American Heart Association and European Society of Thrombosis'. [117-119] There is currently no specific antidote for the new oral anticoagulants (Apixaban, Dabigatran, Rivaroxaban), and no RCT's have been conducted for the treatment of ICH associated with them.

Management of Increased Intracranial Pressure and Brain Edema

Increased ICP may result after mass-effect caused by hematoma or surrounding edema, inflammation and destruction process caused by bleeding, or obstructive hydrocephalus from IVH or hematoma pressing CSF circulation system. [120,121] It is considered to be a major contributor to mortality and poor functional outcome after ICH, and detection & treatment of it, thus, is essential. [121,122]

ICP monitoring is done in all patients with large hemorrhage, IVH, GCS score of 8 or less, those patients whose condition is thought to be deteriorating due to elevated ICP, and those who need to be sedated or pharmacologically restrained and that way have compromised clinical monitoring, those with EVD & obviously those who are treated for increased ICP. [123] Patients with small hematomas and limited IVH usually doesn't require treatment to lower ICP. Management of raised ICP should be directed at the underlying cause, particularly if caused by hydrocephalus or mass effect from the hematoma. [120,121,124,125] We are still lacking evidence from RCTs to make strong recommendations on measures to lower ICP in adults with ICH. [126]

Hyperventilation is commonly used to treat ICP. In case of impending transtentorial herniation, a combination of hyperventilation and osmotherapy is frequently used, though data from RCTs is lacking. [127] Osmotherapy is the first-line medical treatment for increased ICP by reduction of perihematomal edema. It has been proved promising in animal models, and two nonrandomized clinical trials. [127-132] Mannitol can be used, for it is believed to extract water from the cerebral extracellular space of both normal and oedematous brain into the intravascular compartment. Glycerol has been tested in one RCT on 107 ICH patients over 4 hours for 6 consecutive days, but no differences emerged at 6-month mortality or disability in comparison to placebo. [133] Neuromuscular paralysis combined with adequate sedation, as well as optimal head positioning can reduce elevated ICP.

General anaesthesia by barbiturates and induced hypothermia should only be used if other methods are ineffective. [134-137] Pilot data

indicated mild hypothermia (35°C) started within 12 hours from onset for 10 days reduced perihematomal edema and resulted in relatively high survival and functional outcome at 12 months. [137]

Blood Pressure Management

High BP commonly occurs with ICH and usually last for some days before returning to baseline. [138] At presentation, 75% of ICH pts have 'systolic blood pressure' (SBP) >140 mmHg, and 20% have >180 mmHg. [139] High BP is caused by the activation of a combination of neuroendocrine system (catecholamines), renin-angiotensin axis, and glucocorticoid system involved in a nonspecific response to stress, and further worsened by the patients' chronic hypertension. [140] Brainstem-mediated release of catecholamines may be induced by increased ICP. This hypothesized "Cushing reflex" is a protective response aiming to preserve cerebral perfusion. [141] However, high BP on admission is associated with hematoma expansion and poor outcome. [142-143] It may also worsen cerebral edema. The rationale for lowering BP, therefore, is to decrease ongoing bleeding and reduce hematoma expansion.

It has been suggested that overaggressive treatment of BP may, however, decrease CPP and worsen brain injury, particularly if increased ICP is present, for it may hamper autoregulation. [144,145] The absence of ischemic penumbra around a small-volume ICH means that sudden therapeutic reduction of BP does not necessarily involve the same risks that are more common with ischemic stroke. Studies on autoregulation, cerebral perfusion, and one randomized study showed no significant reduction in cerebral blood flow if the pressure is lowered 20% or less in the acute phase. [146-148] One observational study reported better outcomes in ICH patients whose BP was maintained below mean arterial pressure (MAP) of 125 mmHg. [149]

'American Heart Association's guidelines' suggests that, aggressive hypertension management should be implemented if a patient's systolic blood pressure (SBP) is greater than 200 mmHg or their mean arterial blood pressure (MAP) is greater than 150 mmHg. If, on the other hand, a patient's SBP is greater than 180 mmHg or MAP is greater than 130 mmHg and there is no indication or suspicion of elevated ICP, then

modest management should be implemented, with the goal being 160/90 mmHg and MAP of 110 mmHg. While the best time to switch from intravenous to oral antihypertensive medicine is unknown, stable patients are typically advised to switch between 24 and 72 hours later.

Prevention and Management of Seizures

Post-stroke seizure and post-stroke epilepsy may occur as the presenting symptom or complication of haemorrhagic or ischemic stroke. 'Early seizures' are more common in patients with ICH in comparison to other stroke subtypes, and it also associates with lobar lesion location. [150,151] Clinical seizures occur in 2.7 to 25% of patients having ICH, with most of them occurs in the initial period of ICH. [152,153] Seizure occurring after 2 weeks of ICH onset is higher risk for recurrent seizure than one occurring within 2 weeks, and long-term prophylactic anticonvulsant treatment may be needed. Chronic epilepsy develops in 13% of 30-day to 2-year survivors and in 7% of the 2- to 5-year survivors. [152]

The clinical impact of those subclinical seizures detected only in EEG is uncertain. Seizures which develop in the 1st week after ictus are defined as early seizures, and it is seen in around 14% of the patients with ICH.[153] One RCT investigated the adverse effects of diazepam. [154] These investigators reported diazepam is responsible for increased pneumonia and 41 deaths. Investigators from another RCT found that patients receiving valproic acid were less likely to suffer from early seizures, but overall seizure rates by one-year follow-up did not differ.[155] While early seizures do not affect the neurological outcome or mortality at short- or long-term follow-up. Phenytoin use and prophylactic antiepileptic drug (AED) use is associated with poor outcome after ICH. [156-158] In one large single-center study prophylactic AED use significantly reduced clinical seizures in patients with lobar ICH. [159]

AHA guidelines state that only patients with clinical seizures or electrographic seizures accompanied by change in mental status should be treated with AED. Patients with decreased mental status which is not proportionate to the degree of brain injury should be

monitored by continuous EEG. Both AHA and ESO guidelines state that, because of less RCTs, utility and optimal use of AED remains unresolved. [160,161] Intravenous lorazepam in usually the first-line drug for acute management of seizure, and AED treatment should follow the local hospital guidelines.

Blood Sugar Control

Increased blood sugar level is commonly seen after ICH and associated with long term ICU and hospital stay and worsened outcome.[162] Hyperglycemia is considered deleterious to damaged brain, but the exact mechanisms are not known. [163,164] It is commonly seen in patients with ischemic stroke, it is associated with poor outcome, and recommended by AHA and ESO guidelines to be treated with intravenous insulin. [165-167] Hyperglycemia has been reported to worsen the outcome after ICH also with or without diabetes. [168,169]

In patients with ischemic stroke or ICH there is increased use of insulin which may result in hypoglycemia with reduced cerebral extracellular glucose concentration, which also causes increased mortality. [170-172] Rapid decrease in serum glucose level also decreases serum osmolality and may theoretically worsen cerebral edema. Some guidelines recommend lowering glucose levels if exceeding 10.0 mmol/L (180 mg/dl) with avoiding hypoglycemia and large swings in blood glucose concentration.5,200 Glucose containing fluids should be avoided in ICH patients unless they are hypoglycemic. Other systemic medical complications, such as infections and fever, may also cause Hyperglycemia, and should be treated accordingly.

Fluid Management and Electrolyte Imbalance

Electrolyte disturbances may arise after brain injury due to the major role of the CNS on controlling “sodium, potassium, chloride, calcium, magnesium, and water homeostasis” (CSWS, Cerebral Salt Wasting Syndrome), dehydration also cardiac failure. [173-175] Hypo- and hypervolemia both have harmful effects on cerebral perfusion and functioning of other organs. Establishing and maintaining normovolemia is, therefore, the main goal of fluid management in acute ICH. A 15.6% prevalence of hyponatremia among ICH patients has

been reported and identified as a predictor of in-hospital mortality in two recent studies. [176]

Data on other electrolyte disturbances and their effect on ICH prognosis are still limited, but sodium and potassium imbalance have been associated with worse outcomes after ischemic stroke. [177] Rapid increase of sodium concentration may cause fatal pontine myelinolysis, while potassium disturbances may cause cardiac arrhythmias. [178] Isotonic infusion of intravenous fluids without glucose should be used in general to maintain adequate intravascular volume to optimize cerebral perfusion. Hypotonic solutions are contraindicated, because of the risk of increase of brain edema as a consequence of reduced plasma osmolality, and high glucose concentration has adverse effects on injured brain. Electrolyte disturbances should be investigated and corrected according to the severity, suspected duration, and cause. Severe hyperpotassemia may require temporary intravenous glucose and insulin treatment. [178]

Controlling Body Temperature

Central fever and infections may complicate ICH, and fever is an independent predictor of poor outcome in these patients. [179] Body temperature should be, therefore, maintained at normal to near-normal levels. Acetaminophen or cooling blankets are used in treatment of hyperthermia >38.5°C. [180] Hypothermia may be beneficial in some individuals, which acts as anti-inflammatory cytokine, reduces oxidative stress, and is also helpful in improvement of the outcome. [181-183] Due to the lack of RCTs, no strong guidance is available for optimal treatment of fever in patients with ICH. [184] Early treatment with antipyretics may be considered in clinical practice, but preventive treatment is not recommended. Appropriate cultures and smears (tracheal, blood, urine, CSF) should be obtained, and patient physically examined to determine the source of infection in febrile patients and in those at risk for infection, as well as appropriate antibiotics initiated.

Venous Thromboembolism Prophylaxis and Treatment

In the acute phase of ICH, patients are most often immobilized due to neurological deficit or impaired consciousness, putting them at risk for

deep vein thrombosis (DVT), and subsequently for pulmonary embolism (PE). Without any preventative measures DVT develops in up to 50% of ICH patients. [185-187] Measuring D-dimer after 2 weeks from the ictus may be useful to detect DVT. [186,188] As many as 30% of patients with PE show no evidence of lower extremity DVT. [189]

'Thigh-high or Below-knee elastic stockings', 'Intermittent pneumatic compression' (IPC), and anticoagulation by subcutaneous low-molecular-weight heparin (LMWH) and unfractionated heparin have been investigated in multiple RCTs for DVT prevention after ICH. [190-193] Elastic stockings alone are ineffective, while IPC has been proved superior in comparison to other methods and is currently recommended by AHA and ESO both to improve outcome and reduces the chance of developing DVT in immobile patients with ICH. [184]

LMWH seems to be safe initiated on the second day after ICH onset in neurologically stable patients, but may not be more effective than elastic stockings. [194] Inferior vena cava (IVC) filters or 5 to 10-day course of full-dose LMWH followed by 3-months of low-dose LMWH is preferable than vitamin K antagonist for DVT treatment. [195]

Infection Complications

Stroke induces systemic immunosuppression by cellular pathways. [196-197] Infections are frequently complicating the early course and recovery from stroke, and seem to be more often present in patients with ICH than those with ischemic stroke. [198] A meta-analysis of 87 studies involving 137817 patients investigated infections and other complications among stroke patients. [199,200] The key message of these studies is that infections are common, most often include pneumonia and urinary tract infection (UTI), and associate with high death rate, worse functional outcome, and long-term hospital stay. In addition to cellular and biochemical mechanisms of immunosuppression induced by ICH, dysphagia, mechanical ventilation, tracheostomy, and feeding by Ryles tube have also been reported to increase risk for pneumonia. [201-203]

Impaired consciousness is the single most important risk factor for pneumonia. Dysphagia is a frequent symptom of ischemic and

haemorrhagic stroke both, being present in up to 50% of stroke patients, and may cause broncho aspiration with subsequent pneumonia. [204] Pneumonia develops in 14% to 20% of all ICH patients, and associates with increased morbidity, mortality, and length of stay. [203,205,206] UTI complicates the course of ICH in up to 21% of patients. [205] Stroke-induced immunosuppression, bladder dysfunction, use of Foley urinary catheter, and female gender predispose for UTI. [207] Even without a catheter, stroke patients have more than double the odds of developing UTI in comparison to general medical and surgical populations. [208] UTI alone predicts poor outcome, but may also progress into sepsis with fever, and cause delirium, even stronger predictors of increased mortality. [205,209-211] Unnecessary use of Foley catheters should be avoided. New strategies such as condom catheters and antiseptic/antibiotic-covered catheters are under development.

Cardiac Complications

Cardiorespiratory instability usually occurs within 24 hours following ICH onset. [212] Knowledge regarding occurrence, impact, and underlying mechanisms of cardiac complications after ICH, as well as strategies to prevent them is still limited. Severe in-hospital heart related problems include myocardial infarction (MI), heart failure, ventricular arrhythmias, and sudden death due to cardiac arrest.[213] They likely result from brain injury causing disturbance in central autonomic control. Post-stroke cardiac complications result in higher mortality, need for intensive monitoring, and increased length of stay with higher costs. [214-216]

Between 2% to 6% of patients die from a cardiac cause within three months after ischemic stroke, and up to 19% suffer a serious cardiac adverse event. [217,218] MI has been reported occurring in 2% within 3 months after ICH. [219] In-hospital occurrence of MI after ischemic stroke or ICH has been reported being between 1% and 5%. [220] Arrhythmias may precede or be caused by heart failure, which may further lead to neurogenic pulmonary edema. [221] Cardiovascular comorbidities, higher age and stroke severity have been identified to

increase the risk of cardiac morbidity and mortality after stroke. [214-216,222] Troponin elevation, atrial fibrillation, and ischemic ECG changes may be associated with higher in-hospital mortality. [223,224] Both troponin T elevation and ECG changes, however, are frequent after stroke, and most often are unrelated to acute myocardial ischemia. [225,226] Elevated troponin I independently associates with higher in-hospital mortality and cardiac causes of death after ICH, but is present only in 1% of ICH patients. [227]

Takotsubo cardiomyopathy is a phenomenon caused by stress, in which sudden reversible weakening of left ventricle occurs. [228,229] ICH may provoke Takotsubo cardiomyopathy, but the rates have not been investigated. It has been reported to occur in 1% of patients with ischemic stroke, with most occurring during 10 hours after onset, but some even after 12 days, and female gender and insular damage being risk factors. It may cause heart failure, ventricular arrhythmias, and ventricular rupture, all potentially lethal, particularly in vulnerable ICH patients. [230]

Renal Failure

More evidence exists on renal failure being associated with poor outcome after ischemic stroke, but only few studies have investigated renal failure among ICH patients. [231,232] CKD has been reported in as many as one third of ICH patients, and acute renal failure in 7%, more often among women, elderly, and those with other comorbidities, such as diabetes. [231,232]

Chronic kidney disease and renal failure seem to increase mortality by kidney function: the lower the glomerular filtration rate (GFR) is, the higher the mortality rates are. [231,232,233] The proportions of renal failure preceding and appearing during acute ICH, the pathophysiological mechanisms by which ICH affects kidney function, and the strategies of prevention and treatment to address this issue and improve outcome remain unresolved.

Surgical Treatment of ICH

The few basic goals of surgical evacuation of hematoma are:

- To reduce mass effect

- To stop the discharge of ‘neuropathic products’ from hematoma
- To prevent ‘prolonged interaction between the hematoma and the normal tissue’, which can initiate pathologic process.

Some patients have a penumbra of functionally impaired but potentially viable tissue around ICH. Surgical removal of clot may improve the function and recovery in this penumbra.

Various surgical approaches for Spontaneous ICH:

1. Craniotomy with Evacuation of Hematoma
2. Decompressive craniectomy with or without evacuation of hematoma
3. Minimally invasive techniques such as endoscopic hematoma evacuation
   and stereotactic hematoma evacuation
4. Burr hole and EVD for acute hydrocephalus, fibrinolysis of IVH using
   ‘Recombinant tissue plasminogen activator’ (rtPA), or streptokinase are other
   indications for surgery, especially in IVH and intracranial pressure (ICP)
   monitoring.

Craniotomy for Supratentorial haemorrhage

The benefits of clot removal are addressed in two randomized trials. ‘The Surgical Trial in Intracerebral Haemorrhage’ in random 1033 patients with supra-tentorial hemorrhage (lobar or ganglionic hematoma) to early surgery (in less than 96 hours of ictus) vs. standard of care (i.e., medical management with delayed surgery if necessary) [234]. There was no any difference in favourable functional outcome at 6 months found (p =0.414). However, few patients with superficial ICHs (lobar haemorrhage lying in less than 1 cm of the cortical surface) underwent surgery and had better outcomes. This result prompted a second trial, STICH I, aiming at randomizing patients with superficial lobar hematomas (10-100 ml) to early surgery versus medical management with delayed surgery if necessary [235]. Patients with IVH or coma were excluded.

Early surgery did not significantly alter death or severe disability in STICH II. Notably, early surgery increased the likelihood of a favourable result over conservative treatment at enrolment for patients whose prognosis was projected to be poor. Even in the patient group whose prognosis was projected to be good at enrolment, there was no discernible benefit for patients undergoing early surgery. [235]

Decompressive Craniectomy for Supratentorial ICH

There hasn't been much research done on how Decompressive Craniectomy (DC) might help people with ICH. Based on the findings of the initial STICH trial, a number of writers have hypothesized that DC may be able to improve outcomes for a subset of patients who have high ICP and ICH-related mass effect. [236-238] Patients with considerable midline shift, large hematomas or refractory intracranial pressure were more likely to be in a coma (GCS score <8) and to be included in the study.

One study of DC without evacuation of hematoma matched 12 consecutive patients with supratentorial ICH to control subjects via propensity score. [236] Median pre-operative GCS score and volume of hematoma was 8 and 61.3cc respectively. Three patients in the study group died compared to 8 in the control group, whereas 9 patients had good outcome. Another study on DC without evacuation of hematoma included 5 patients with recalcitrant elevated ICP. [238] This small cohort fared better than matched control subjects from the author's institutional prospective ICH database. A systematic review of studies in which DC was performed in the setting of spontaneous ICH suggested that DC with hematoma evacuation might be beneficial and might improve outcomes.[237]

The use of hematoma evacuation by craniotomy and DC was also studied. In retrospective research, “Hayes et al.” compared DC and hematoma evacuation. A comparison was made between 10 patients who received hematoma evacuation with DC and 16 patients who underwent hematoma evacuation by craniotomy in the subgroup of patients with Putaminal hemorrhage. Individuals undergoing

decompressive craniectomy had a higher probability of having a lower preoperative GCS (GCS < 8, p = 0.019).

DC have a tendency toward improved outcome and a notable improvement in midline shift in cases of Putaminal hemorrhages. Eight individuals in the subgroup of patients with lobar ICH had hematoma drainage combined with a decompressive craniectomy, while 17 patients had hematoma drainage alone through a craniotomy. Individuals undergoing decompressive craniectomy exhibited a higher likelihood of both large midline shift (p=0.022) and right-sided hemorrhage (p = 0.011). Decompressive craniectomy did not prove to be beneficial for this subset of individuals who had lobar hemorrhages. [239]

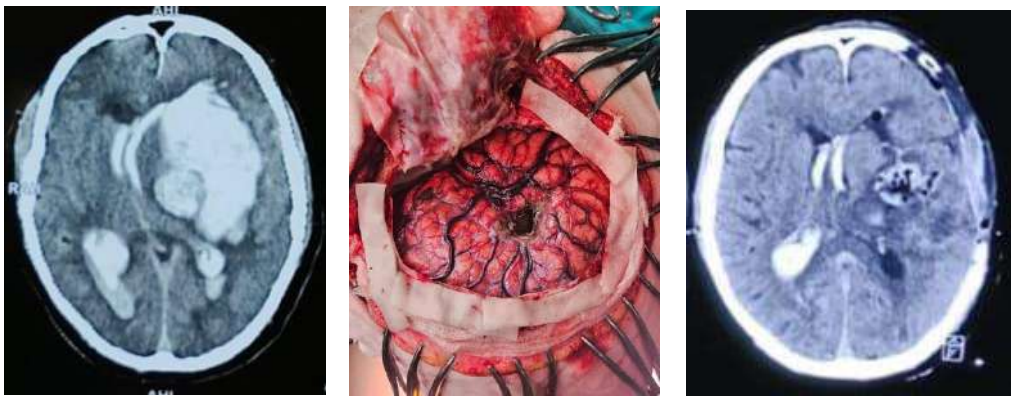


Photo 2: Shows Pre-op CT scan, Intra-op picture and Post-op CT scan of Large Left Gangliocapsular ICH with Intraventricular extension.

Posterior fossa haemorrhage

'The posterior fossa haemorrhage', which includes the cerebellum and brainstem, is frequently the site of life-threatening issues such as acute hydrocephalus resulting from compression of the fourth ventricle, direct brainstem compression, and/or herniation via the foramen magnum. Conservative care, EVD insertion, or posterior fossa (sub-occipital) decompressive craniectomy were among the available treatment options. Because no RCT's has been conducted to determine the optimal strategy or time to treat infratentorial hemorrhage, the existing evidence is based on class III research.

Different protocols and algorithms were published, directing management strategies based on Gocs and hematoma size, [240] degree of fourth-ventricle compression [241] or GCS and presence of hydrocephalus. [242] Patients with GCS score of 14 or 15 and small sized hematomas less than or equal to 3 mm can be managed medically. Hematoma drainage, either with or without a craniectomy, should be carefully evaluated in cases of neurological impairment. A formal neurological determination of death should be taken into consideration for comatose individuals who lack brainstem reflexes. Emergency hematoma drainage and sub-occipital decompressive craniectomy should be explored for comatose individuals who retain brainstem reflexes. The AHA guidelines do not support the use of External Ventricular Drainage alone in the treatment of cerebellar occupying lesions due to the theoretical risk of upward herniation. [243] This approach is still contentious. Sub-occipital decompressive craniectomy is less necessary when EVD is used alone, according to the management of a recent cohort of patients with cerebellar infarcts.

'Minimally Invasive Surgical Approaches' for ICH

'Minimally invasive surgery' (MIS) is another option and offers some advantages over conventional craniotomy, including the possibility for local anaesthesia, reduced operating time, and reduced tissue trauma. [235,244] Studies on endoscopic clot evacuation have reported decreased mortality, but results concerning functional outcome have been contradictory. [245,246] Stereotactic aspiration and thrombolysis of hematoma by image guidance is the second main type of MIS. [247-250] Preliminary results from "Minimally Invasive Surgery Thrombolysis Plus Recombinant Tissue-type Plasminogen Activator for ICH Evacuation" (MISTIE) and results from "Stereotactic treatment of intracerebral hematoma by means of plasminogen activator" (SICHPA), showed greater clot resolution and decreased perihematomal edema with MIS and rtPA than with traditional medical management. [251,252] According to a recent meta-analysis of 12 RCTs with 1955 patients, those with supratentorial hemorrhage may benefit from MIS rather than conservative medical treatment and conventional craniotomy,

particularly those of age between 30 and 80, those with superficial hematoma, GCS score ≥9, hematoma volume between 25 and 40 mL, and intervention performed in less than 72 hours after onset of symptoms. [253] These findings are supported by another recent meta-analysis comparing stereotactic aspiration and craniotomy. [254]

According to AHA guidelines, last released in 2010, before above-mentioned meta-analyses, the effectiveness of 'minimally invasive' clot evacuation utilizing either endoscopic or stereotactic aspiration with or without thrombolytic usage is uncertain due to greatly depending on the accurate catheter placement and, therefore, considered investigational. The MISTICH and ICES trials are RCTs currently in progress to define whether MIS could improve the prognosis of ICH in comparison to craniotomy, and ECMOH is a RCT investigating whether endoscopic surgery has more advantages than conservative treatment for patients with moderate-volume hematomas in the basal ganglia. [255,256]

Treatment of Intraventricular Hemorrhage and Hydrocephalus

If ICH extends near to ventricles, i.e. originating from basal ganglia or being lobar with large volume, it may spread to CSF circulation (IVH). [257] It is seen among 40% to 60% of all ICH patients. Delayed IVH seems to be less common and more benign than one discovered on admission. [258]

The adverse effect may be related to IVH interfering with normal functions of CSF by causing localized lactic acidosis, and causing mass-effect on periventricular structures, which is associated with global hypoperfusion of overlying cortex. [259] IVH blood clot may also cause obstruction of the CSF flow resulting in obstructive hydrocephalus, and even subsequent herniation, another predictor of poor outcome after ICH, usually manifesting with unconsciousness. [260-262] In addition, it may cause inflammation of the choroid plexus leading to increased CSF production and that way worsening hydrocephalus, as well as cerebral vasospasm. [261] Delayed communicating hydrocephalus may also follow IVH, when the degradation products of the blood clot generate an inflammatory response that gradually occludes the arachnoid granulations where CSF is absorbed. [263]

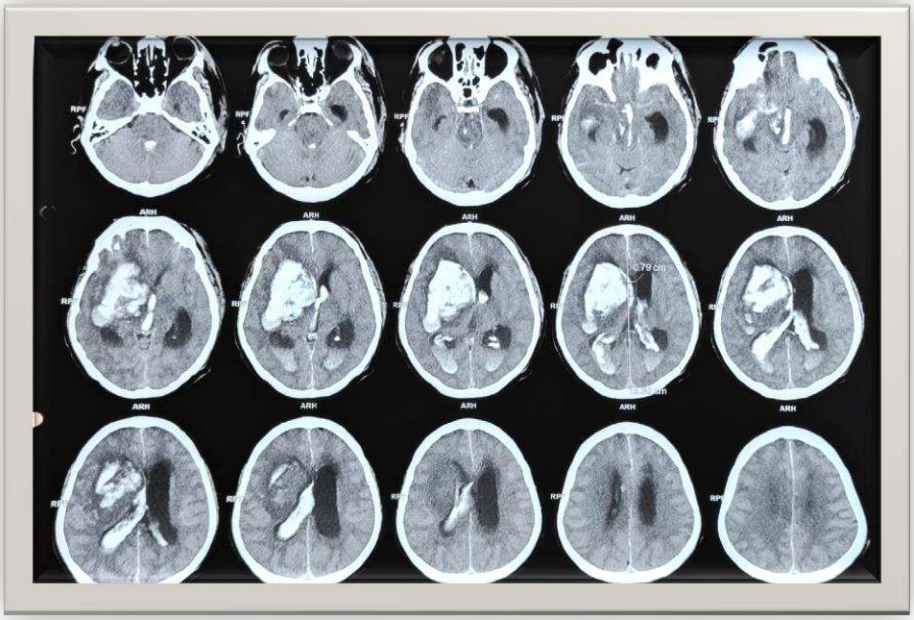


Photo 3: ICH and IVH causing Obstructive Hydrocephalus

Hydrocephalus is a life-threatening condition, and should be treated with EVD of CSF through an intraventricular catheter, as an emergency measure, if necessary. [264,265] EVD reduces ICP, but also carries a risk of clotting and infection. [266,267] Intrathecal thrombolysis with urokinase is used to prevent and treat hydrocephalus, since it shows reduction in death rate and it seems acceptably safe. [267,268-270]

In accordance, a recent meta-analysis concluded intraventricular fibrinolysis being safe, and possibly reducing mortality, improving functional outcome, and diminishing the need for permanent shunting without increasing the risk of infection or rebleeding. [271] Neuroendoscopic removal of blood clot in IVH has shown promising results, but no conclusive evidence yet exists regarding the preference between that and EVD. [272-275]

8. Long-Term Outcome of Intracerebral Haemorrhage

Mortality

As mentioned earlier, ICH is the most catastrophic subtype of strokes, with one-month mortality of 40%, and great proportion of the survivors left with functional impairment. [276-278] Among the one-month survivors

of ICH, risk for long-term mortality is highest in the first year following: 4.5-fold fold the risk in people from the general population. [279] An average annual risk of death of 9.1% has been reported for long-term mortality among one-month survivors of stroke. [280] According to two recent meta-analyses, one-year mortality was 54%, including patients dying within the first 30 days. [281] Five-year and 10-year mortality reached up to 71% and 82%, respectively. [281,282]

Long-term mortality among stroke patients is mainly due to 'cardiovascular diseases', but also cancer, other diseases, accidents, and suicide are other significant causes of death in ICH patients. [280] Higher age, male gender, diabetes, and heart failure also elevates the risk of long-term mortality after ICH. [280,283-285] Studies on ischemic stroke in young adults has shown that young population having ischemic stroke have lower mortality, 7.9% at five years among one-month survivors, lower than their elder counterparts, but higher than general population. [286-289]

Stroke Recurrence

Annual rate of ICH recurrence ranges from 1.3% to 7.4% by several studies. [290-293] Investigators of one, relatively large, population-based study reported cumulative 5- and 10-year rates of recurrence of 9.6% and 14.2%, respectively. [292] Age ≥65 years, lobar hematoma location, and previous ischemic stroke increases the risk of recurrence in multiple reports. [294,295] Lobar location is the most important risk factor with a 3.8-fold increased risk of recurrence. [295] Hypertension is associated with recurrence in some studies. [294,296] IVH, OAC use, diuretic monotherapy, α- or β-blocker monotherapy, poor functional outcome after initial ICH, basal ganglion location of hematoma, and absence of hyperlipidaemia, however, have been reported associating with recurrence in single study. [293,294]

Diabetes is a risk factor known to increase risk for recurrent ischemic stroke, and a tendency with recurrent ICH was reported by one study. [292,297] Among those with Hypertensive ICH, initial and recurrent hemorrhages most often have different site of bleeding. [296] Between 24% and 70% of the recurrent ICH are fatal. [294] Patients with ICH have

even higher risk for ischemic stroke than ICH recurrence. [291,295] Annual occurrence of vascular events in total, including all stroke subtypes and MI, is almost three-fold to ICH-recurrence, with higher age and male gender identified as predictors. [298]

Rehabilitation and Recovery

Neuronal damage caused by ICH results in symptoms and subsequent disabilities depending on the location and magnitude of the brain injury. These disabilities may often present as one-sided motor paresis of upper, lower, or both extremities, paraesthesia, dysarthria, dysphasia, and visual impairment. Other symptoms include neglect, vertigo, headache, central poststroke pain (CPSP), impaired memory, and other neuropsychological or cognitive handicaps. [299]

A recent study reported better survival and functional outcomes in patients with early rehabilitation which was initiated in less than 48 hours following ICH. [300] Initial evaluation by 'physical therapist, occupational therapist, speech therapist, and neuropsychologist' is of grave importance, and intensive and well-organized multidisciplinary rehabilitation should begin in ICSU, since it has been shown to improve survival, recovery, and returning home. [301,302] Early mobilization has been proposed to decrease complications, but avoiding falls, another complication of stroke, is also important. [303] Once the patient is stable, no evidence of neurological deterioration is observed for several days, and patient's condition improves, rehabilitation may be continued either as outpatient or in a rehabilitation center. Early supported discharge and home-based rehabilitation have shown to be cost-effective and to produce comparable results to conventional outpatient rehabilitation. [304,305]

Recovery is more rapid in the first weeks, but may continue for many months after ICH. Speed and grade of recovery are individual, and no hard rule exists on when recovery is over. [306] Cognition, mood, motivation, and social support all are important for recovery. Since the nature of poststroke disability is complex and includes physical and mental impediments, rehabilitation programs should be individually tailored and to enable lifestyle changes. [306] Recovery after ICH can be

improved through progress in the field of neurorehabilitation. The disability caused by ICH is usually a complex combination of physical and mental impediments. Their nature is individual, and depends on the size, location, and severity of the brain tissue damage caused by hematoma. The pre-ICH condition and comorbidities likely affect also. Although data on functional outcome at long-term after ICH has been reported by some studies, but the knowledge regarding it remains limited. Glasgow Outcome Scale, mRS, and Barthel Index have commonly been used to measure the functional outcome.

9. Secondary prevention

The most important etiologic factor to control after ICH is hypertension; however, no ICH specific secondary preventive medications exist. A large proportion of ICH patients are using or have an indication to antiplatelet or anticoagulants drugs. [307] On top of recurrent ICH, ICH survivors also have a risk for thrombotic events, such as ischemic stroke and myocardial infarction, which raises concerns for the benefits and risks of secondary prevention, such as antiplatelet therapy. Especially patients with deep ICH as well as older patients have been noted to have a high risk for future ischemic events. There are currently no RCT's on resumption of OAC after ICH, however, several large observational studies and meta-analyses exist. [308,309] A meta-analysis of ICH patients with atrial fibrillation (AF) showed that the rate of ischemic stroke exceeded the rate of recurrent ICH and anticoagulation reduced the rate of thromboembolic complications and mortality, without causing an increase in recurrent ICH rates. [310] In a study observing patients with or without resumption of antiplatelet after mild or moderate ICH, there was no significant difference in functional outcome or health-related quality of life at 90 days. [311] In light of current evidence, OAC resumption is considered beneficial in decreasing thromboembolic complications and mortality. The resumption may be safer in non-lobar than lobar ICH and should be combined with strict blood pressure control decided individually weighing potential risks and benefits.

A meta-analysis of 8 studies with 6259 patients demonstrated an over representation of cerebellar ICH and IVH among OAC-treated ICH patients and the authors conclude that the mechanisms behind the underlying arteriopathy, pathophysiology and bleeding pattern of OAC related ICH should be investigated more thoroughly.[312]

MATERIALS AND METHODS

SOURCE OF DATA:

The patients presenting to Dayanand Medical College and Hospital, Ludhiana, with Hypertensive Supratentorial Intracerebral Haemorrhage and got operated will be taken into study group. Study was conducted from 1st December 2022 to 30th November, 2023.

METHOD OF COLLECTION OF DATA:

- All the patients more than 18 years was evaluated neurologically and radiologically with non-contrast CT of head.
- Detailed clinical and neurological examinations was performed in all patients
- All relevant information like Demographic (Age, Sex, Address), Family history, Personal History Co-morbidity etc. was collected

INCLUSION CRITERIA:

- All patients above 18 years of age with Supratentorial Hypertensive intracerebral haemorrhage who underwent surgical intervention were taken into study.

EXCLUSION CRITERIA:

- Head trauma
- Intracranial neoplasm
- Aneurysm
- AVM
- Bleeding diathesis
- Infratentorial haemorrhage
- Not willing for surgery

DURATION OF STUDY:

This Study was conducted from 1st December 2022 to 30th November 2023 in Department of Neurosurgery, Dayanand Medical college, Ludhiana.

MANAGEMENT PLAN:

Patients was managed according to the clinical and radiological findings and appropriate Surgical procedure was choosen (Craniotomy and hematoma evacuation, Decompressive Craniectomy and

hematoma evacuation, EVD insertion with ICH evacuation, EVD insertion). ICU care and ventilator and ionotropic support was given as required.

Indications for Surgery:

1. Patients with Supratentorial ICH of size ≥ 2 cm of diameter and with GCS of ≥ 5. [313]
2. Spontaneous Lobar ICH on CT scan (≤ 1cm from the cortical surface of the brain) [314]
3. Temporal Hematoma as small as 30ml [315]
4. Hematoma occupying 4-8% of intracranial space with clinical status of the patient. [315]
5. Hematoma occupying 8-12% of intracranial space [315]
6. Hematoma volume of 50-100ml with evidence of raised ICP (Pupillary asymetry, and drop in GCS)

OUTCOME AND FOLLOW UP:-

Outcome was assessed by using GOS (Glasgow outcome scale), GCS (Glasgow coma scale) at the time of discharge and on follow up at 2 months and mRS (Modified Rankin scale) at the time of discharge and on follow up at 3 months.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

This prospective study was conducted during the time period from December 2022 to November 2023 in the Department of Neurosurgery, Dayanand Medical college and Hospital, Ludhiana, after obtaining clearance from hospital ethics committee. This follow-up study included 50 patients of either sex with diagnosis of Hypertensive Supratentorial ICH and operated for the same by different surgical interventions. Patients were assessed as per History, Examination, Radiological study and by various scoring system included in the protocol. Patients were operated and GCS, GOS, mRS at discharge and at 2 months were done to analyze the clinical outcome. The data was collected, analyzed and the results are as follows:

Table 1) Age distribution of cases studied.

Age group (years)	No. of cases	% of cases
21 – 30	1	2.0
31 – 40	7	14.0
41 – 50	13	26.0
51 – 60	14	28.0
61 – 70	11	22.0
71 – 80	2	4.0
81 – 90	2	4.0
Total	**50**	**100.0**

Table 1 and Figure 1 shows that the age distribution of cases studied. The total number of cases included were 50. The youngest patient in the study was of 30 years and the oldest patient was of 87 years old. The average age was 53.58 years (median 52.0 years) with standard deviation (SD) was 12.92 years.

Figure 1) Age distribution of cases studied

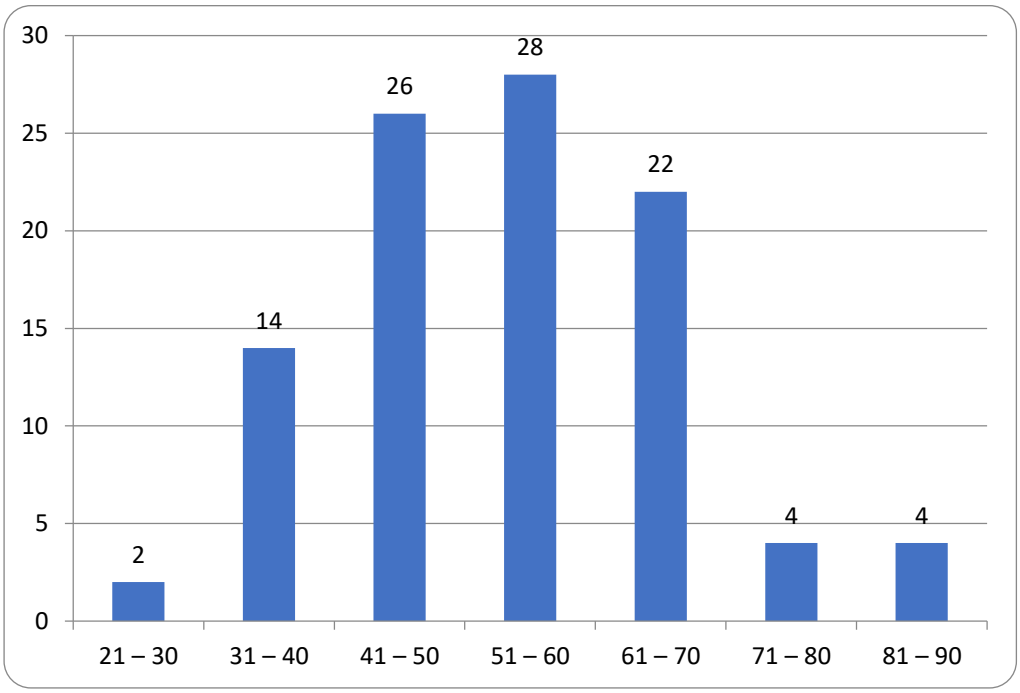


Table 2) Gender distribution of cases studied.

Gender	No. of cases	% of cases
Male	42	84.0
Female	8	16.0
Total	**50**	**100.0**

Table 2 and Figure 2 shows that the gender distribution of cases studied. Out of 50 cases, 42 (84.0%) were male and 8 cases (16.0%) were female. Gender distribution showed male predominance in the study.

Figure 2) Gender distribution of cases studied

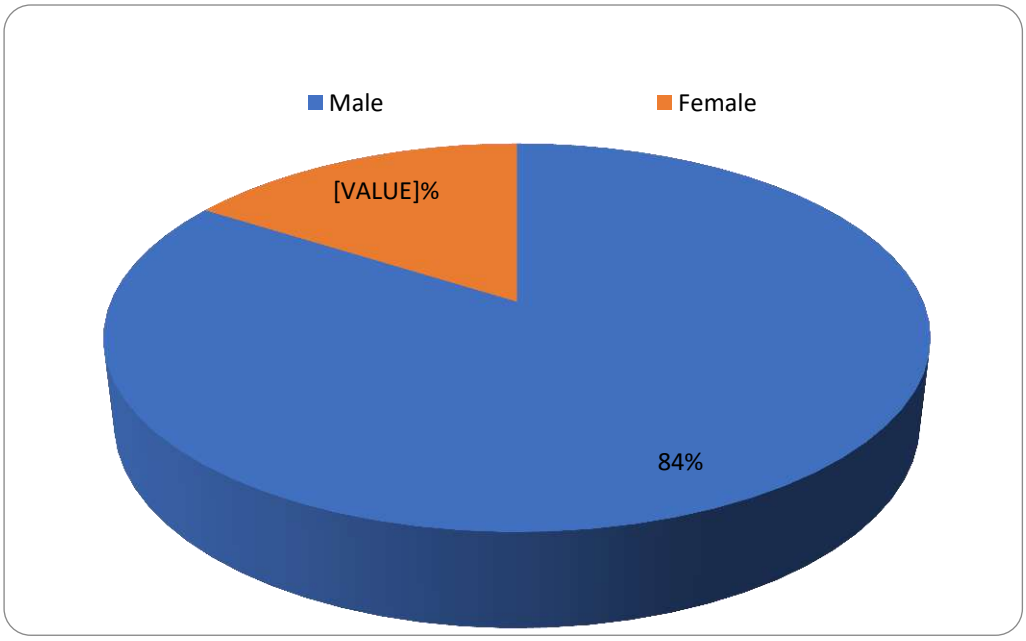


Table 3) Distribution of cases studied according to symptoms.

	Present		Absent	
Symptoms	**No. of cases**	**% of cases**	**No. of cases**	**% of cases**
Headache	8	16.0	42	84.0
Vomiting	11	22.0	39	78.0
Seizures	3	6.0	47	94.0
Hemiparesis	42	84.0	8	16.0
LOC	9	18.0	41	82.0
Altered sensorium	47	94.0	3	6.0
Aphasia	7	14.0	43	86.0
Blurring of vision	1	2.0	49	98.0

Table 3 and figure 3 shows the distribution of symptoms among the cases studied. Altered sensorium was the most common presenting symptom which was present in 47 cases (94.0%), hemiparesis was the second most common symptom which was present in 42 cases (84.0%), the third most common symptom was vomiting which was seen in 11 cases (22.0%), Headache was present in 8 cases (16.0%), 9 cases (18.0%) had loss of consciousness, 7 cases (14.0%) had aphasia, 3 cases (6.0%) had seizures and only 1 case (2.0%) had blurring of vision. Symptoms were non-mutually-exclusive, as a single patient would have multiple symptoms.

Figure 3) Distribution of symptoms

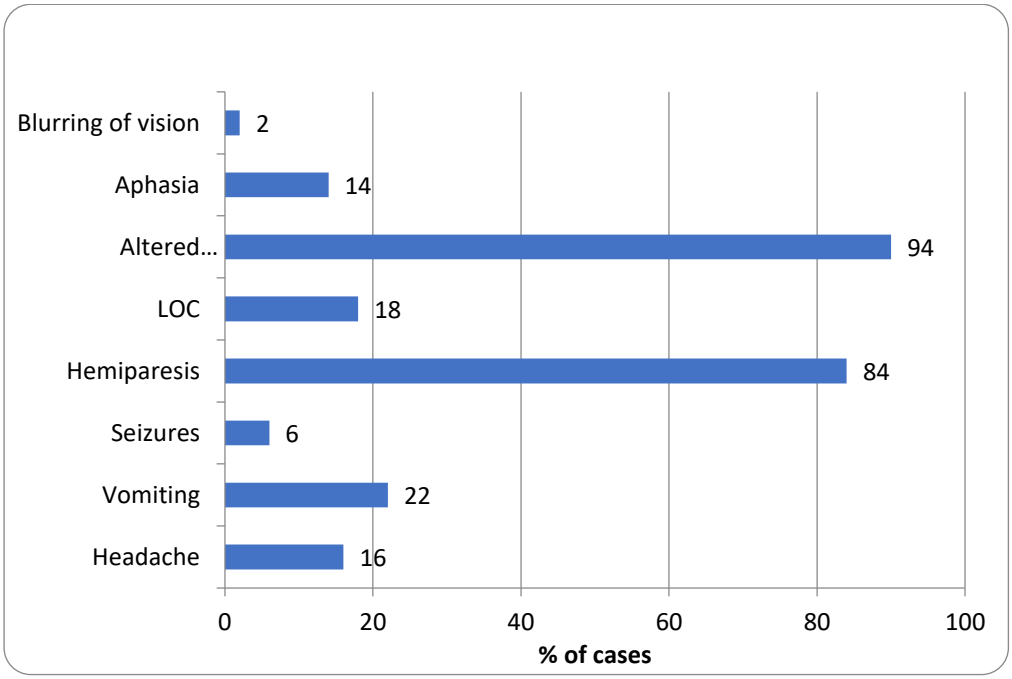


Table 4) Distribution of cases according to GCS (Glasgow coma scale) score on admission.

GCS on admission	No. of cases	% of cases
3 – 5	19	38.0
6 – 8	16	32.0
9 – 11	12	24.0
12 – 15	3	6.0
Total	**50**	**100.0**

Table 4 and Figure 4 shows the distribution of GCS on admission among the cases studied. Out of 50 cases, 19 (38.0%) had GCS between 3 – 5, 16 cases (32.0%) had GCS between 6 – 8, 12 cases (24.0%) had GCS between 9 – 11 and 3 cases (6.0%) had GCS between 12 – 15 on admission. Majority of cases had GCS between 3 – 5 in the study.

Figure 4) Distribution of GCS on admission

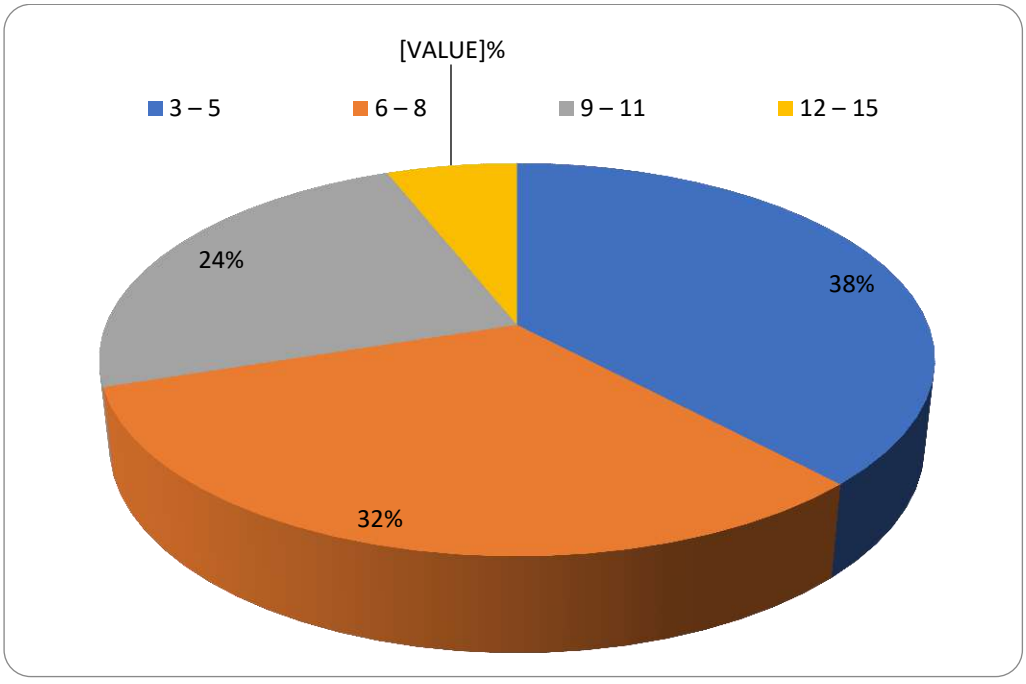


Table 5) Distribution of cases according to status of Pupil asymmetry (Anisocoria).

Pupil asymmetry	No. of cases	% of cases
Yes	13	26.0
No	37	74.0
Total	**50**	**100.0**

Table 5 and Figure 5 shows the distribution of status of pupil asymmetry among the cases studied. Out of 50 cases, 13 cases (26.0%) had pupil asymmetry and 37 case (74.0%) were without pupil asymmetry in the study.

Figure 5) Distribution of pupil asymmetry

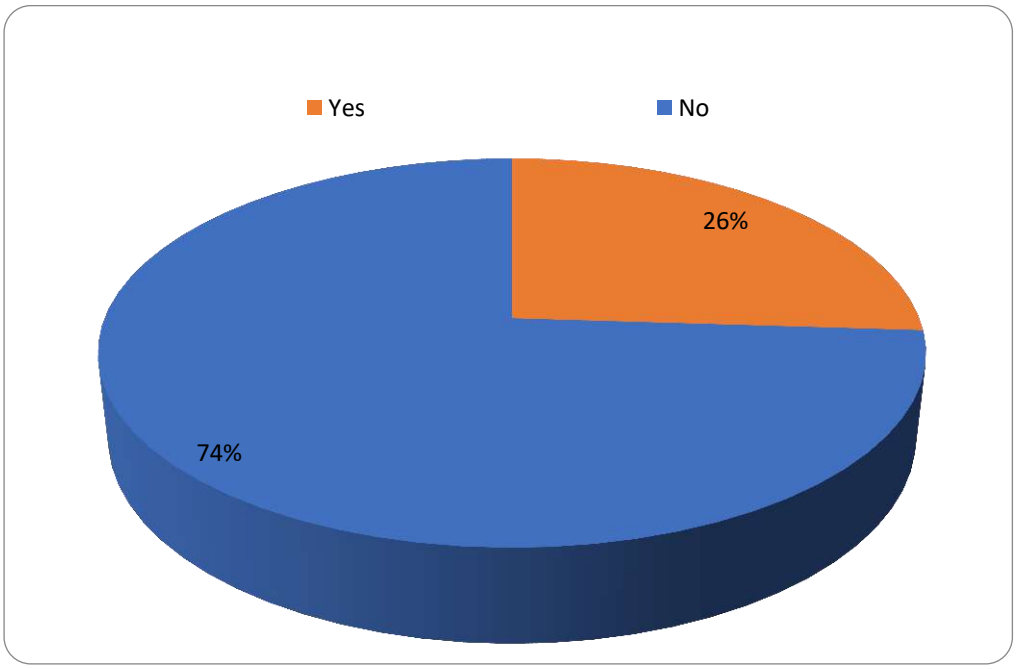


Table 6) Distribution of cases studied according to co-morbidity.

	Present		Absent	
Co-morbidity	**No. of cases**	**% of cases**	**No. of cases**	**% of cases**
Hypertension	38	76.0	12	24.0
Diabetes mellitus	8	16.0	42	84.0
Coronary artery disease	3	6.0	47	94.0
Chronic kidney disease	4	8.0	46	92.0
Hypothyroidism	4	8.0	46	92.0

Table 6 and figure 6 shows the distribution of co-morbidity among the cases studied. There were 38 cases (76.0%) who were hypertensive, 8 cases (16.0%) had diabetes mellitus, 3 cases (6.0%) had CAD, 4 cases (8.0%) had CKD, 4 cases (8.0%) had hypothyroidism.

Co-morbidity was non-mutually exclusive, as a single patient would have multiple co-morbidities.

Figure 6) Distribution of co-morbidity

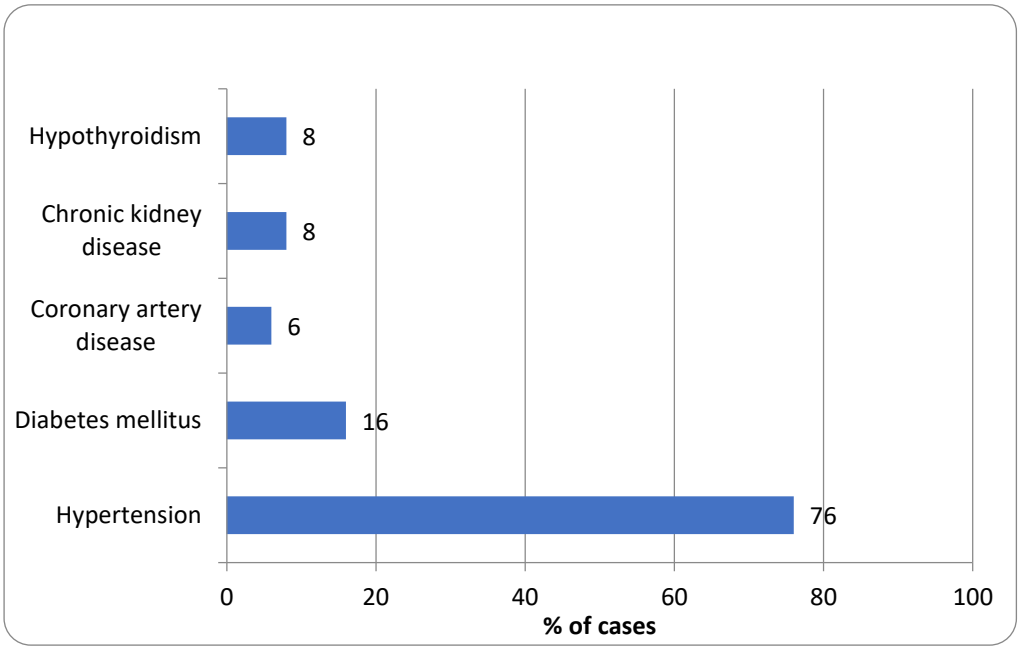


Table 7) Distribution of cases studied according to substance-abuse.

	Present		Absent	
Substance abuse	**No. of cases**	**% of cases**	**No. of cases**	**% of cases**
Alcohol	22	44.0	28	56.0
Smoking	4	8.0	46	92.0
Drug abuse	6	12.0	44	88.0

Table 7 and figure 7 shows the distribution of substance abuse among the cases studied. In terms of substance abuse, 22 cases (44.0%) had alcohol habit, 4 cases (8.0%) were smokers and 6 cases (12.0%) had drug abuse.

Substance abuse was non-mutually exclusive, as a single patient would have multiple habits.

Figure 7) Distribution of substance abuse

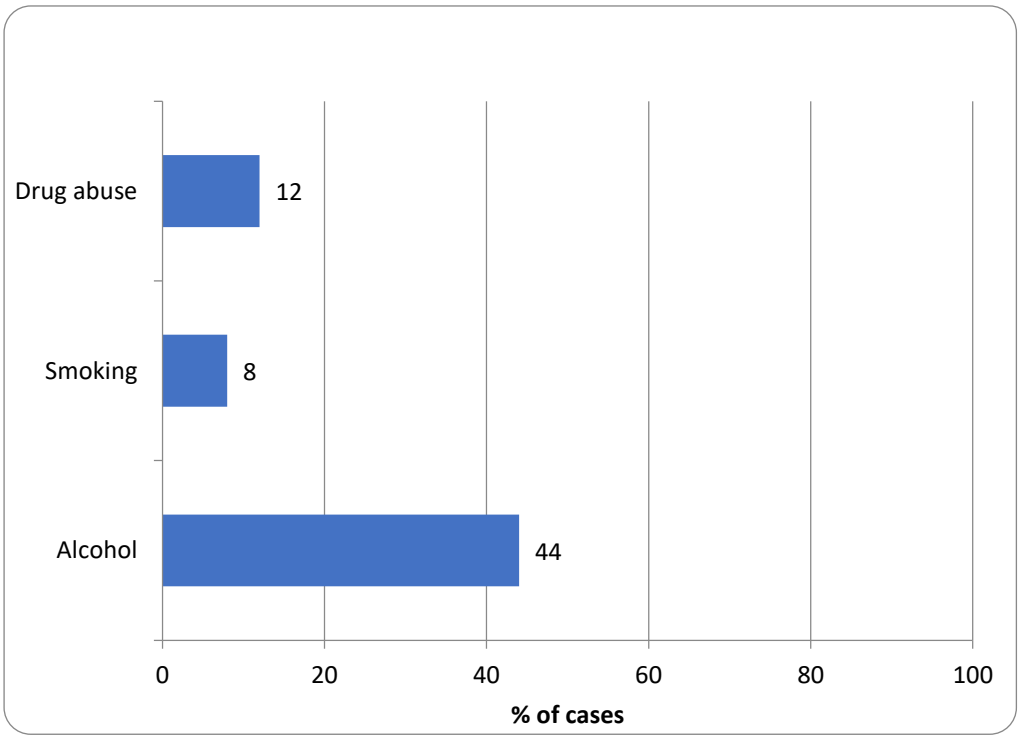


Table 8) Distribution of cases according to location of haemorrhage.

Location	No. of cases	% of cases
Let Frontotemporal	1	2.0
Left Gangliocapsular	15	30.0
Left Thalamic	10	20.0
Left Parietal	1	2.0
Left Temporoparietal	1	2.0
Right Gangliocapsular	17	34.0
Right Thalamic	5	10.0
Total	50	100.0

Table 8 and Figure 8 shows the distribution of location of haemorrhage. Right gangliocapsular region was the most common location of haemorrhage presented with 17 cases (34.0%), 15 cases (30.0%) Left gangliocapsular region involved, 10 cases (20.0%) had left thalamic region, 1 case (2.0%) had left parietal, 1 case (2.0%) had left temporoparietal region and 5 cases (10.0%) had right thalamic region involved in the study group.

Figure 8) Distribution of location of haemorrhage

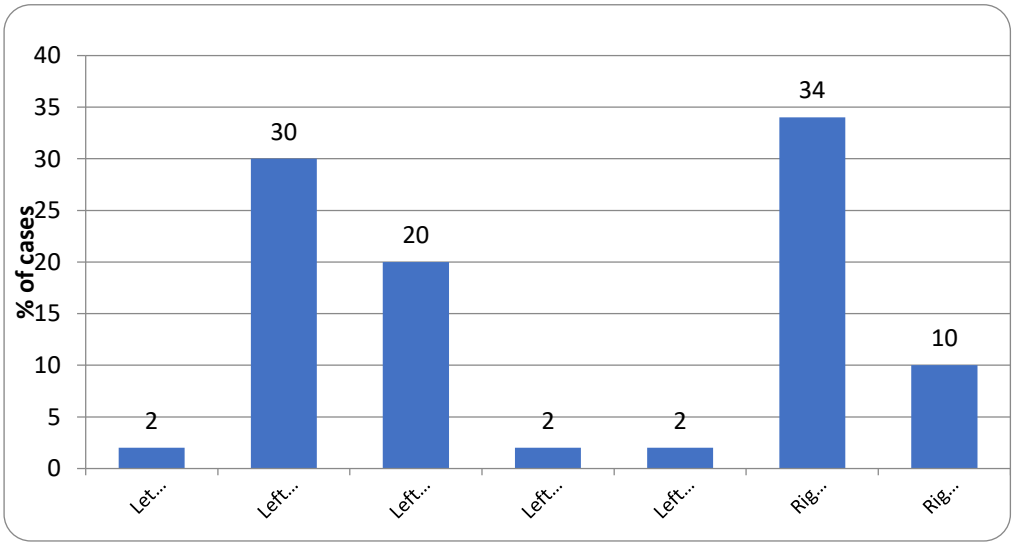


Table 9) Distribution of cases according to volume of bleed.

Volume of bleed (ml)	No. of cases	% of cases
<30	18	36.0
30 – 50	16	32.0
51 – 80	13	26.0
>80	3	6.0
Total	**50**	**100.0**

Table 9 and Figure 9 shows the distribution of volume of bleed among the cases studied. Out of 50 cases, 18 cases (36.0%) had volume below 30 ml, 16 cases (32.0%) had volume of bleed between 30 – 50 ml, 13 cases (26.0%) had volume between 51 – 80 ml and 3 cases (6.0%) had volume of bleed above 80 ml in the study. Majority of cases had bleed volume below 30 ml.

Figure 9) Distribution of volume of bleed

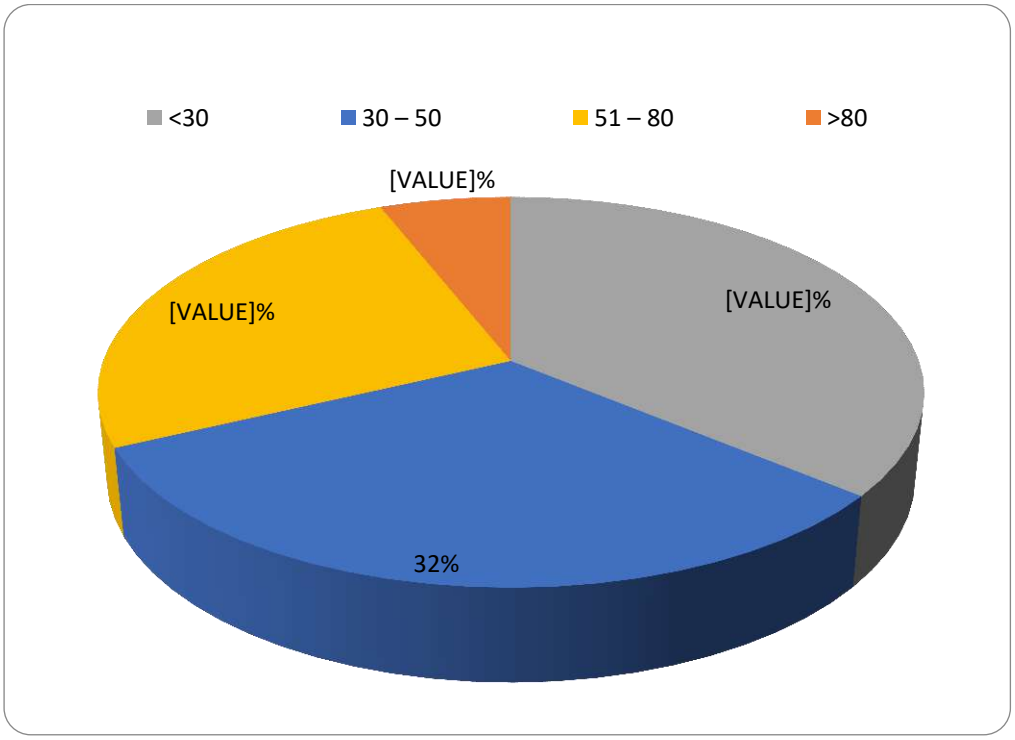


Table 10) Distribution of cases according to mid-line shift.

Mild-line shift (mm)	No. of cases	% of cases
<5 mm	4	8.0
5 – 10 mm	34	68.0
>10 mm	12	24.0
Total	**50**	**100.0**

Table 10 and Figure 10 shows the distribution of mid-line shift among the cases studied. Out of 50 cases, 4 cases (16.0%) had mid-line shift below 5 mm, 34 cases (68.0%) had mid-line shift between 5 – 10 mm and 12 cases (24.0%) had mid-line shift above 10 mm.

Figure 10) Distribution of mid-line shift

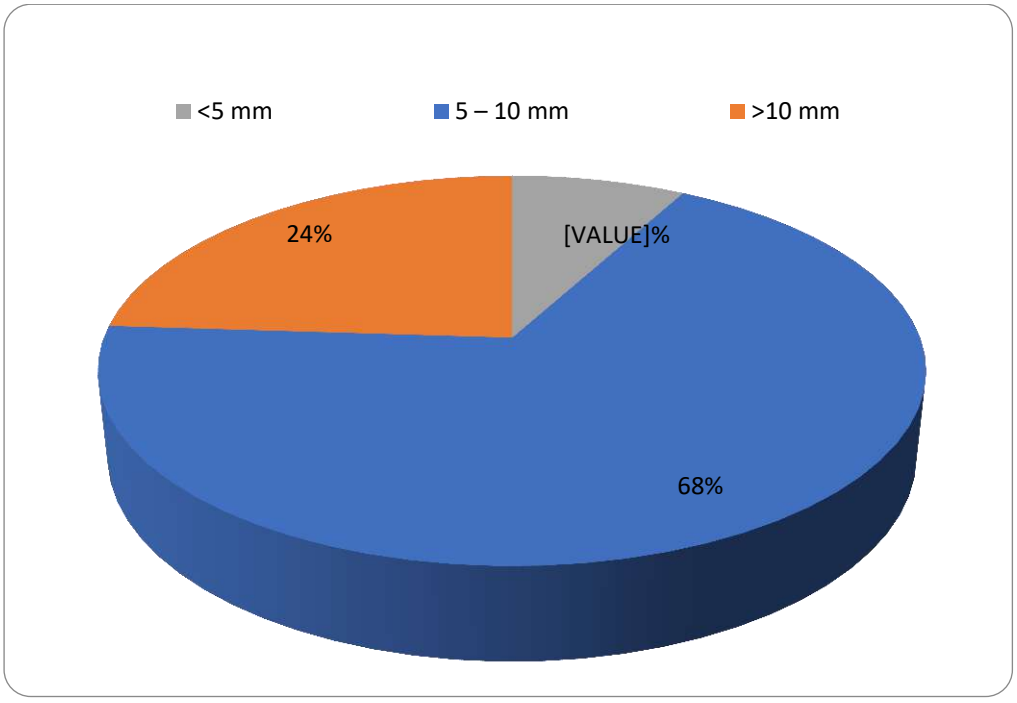


Table 11) Distribution of cases according to associated IVH.

Associated IVH	No. of cases	% of cases
Yes	35	70.0
No	15	30.0
Total	**50**	**100.0**

Table 11 and Figure 11 shows the distribution of status of associated IVH among the cases studied. Out of 50 cases, 35 cases (70.0%) had associated IVH and 15 cases (30.0%) did not have associated IVH in the study group.

Figure 11) Distribution of associated IVH

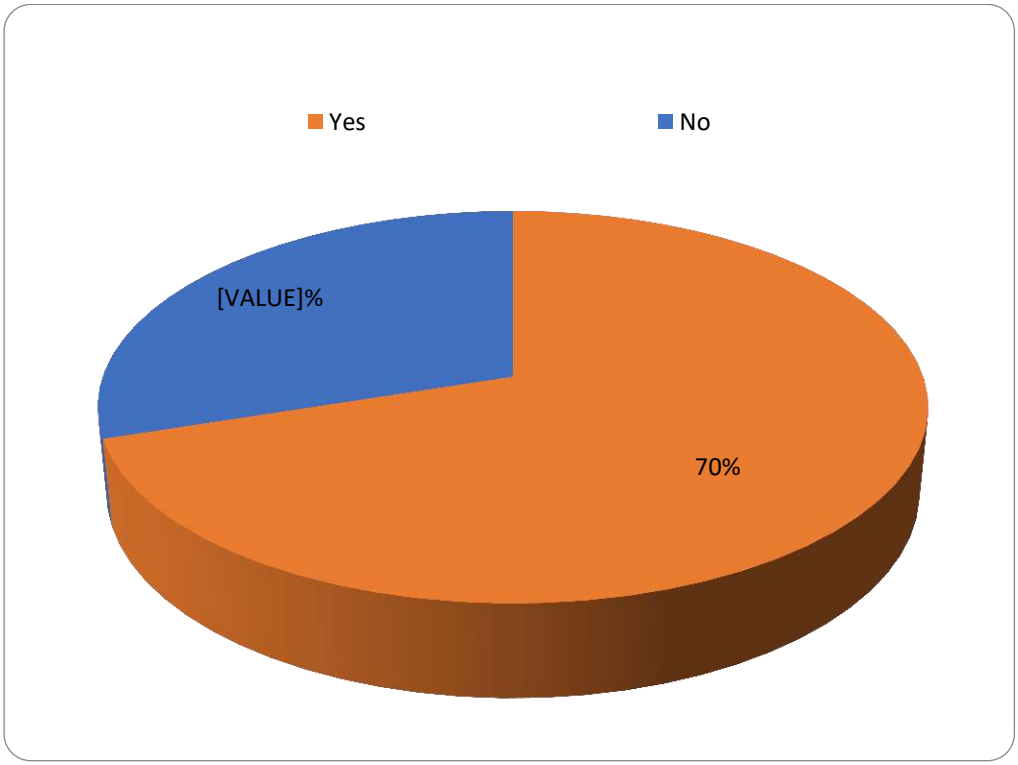


Table 12) Distribution of cases according to operative technique.

Operative technique	No. of cases	% of cases
Craniotomy and hematoma evacuation	3	6.0
Decompressive craniotomy and hematoma evacuation	28	56.0
EVD insertion	16	32.0
EVD insertion with hematoma evacuation	3	6.0
Total	**50**	**100.0**

Table 12 and Figure 12 shows the distribution of operative technique used among the cases studied. Out of 50 cases, 3 cases (6.0%) had Craniotomy and hematoma evacuation, 28 cases (56.0%) had Decompressive craniotomy (DC) and hematoma evacuation and 16 cases (32.0%) had EVD insertion and 3 cases (6.0%) had EVD insertion with hematoma evacuation in the study group.

Figure 12) Distribution of operative technique

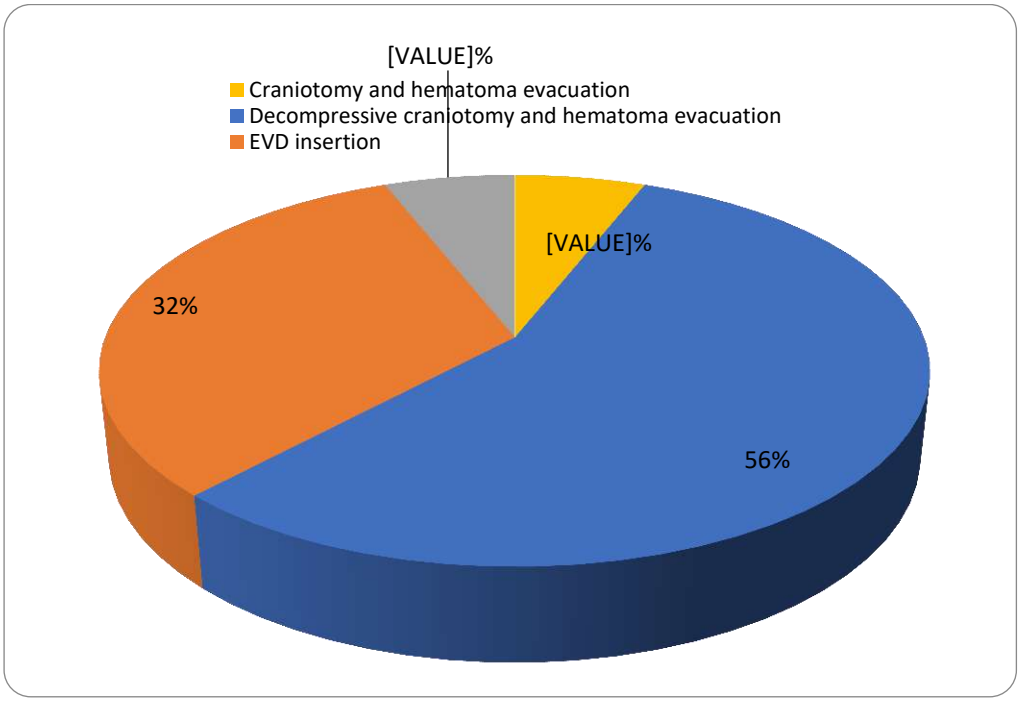


Table 13) Distribution of cases according to GCS on discharge.

GCS on discharge	No. of cases	% of cases
3 – 5	3	6.0
6 – 8	9	18.0
9 – 11	17	34.0
12 – 14	14	28.0
15	5	10.0
Expired	2	4.0
Total	**50**	**100.0**

Table 13 and Figure 13 shows the distribution of GCS on discharge among the cases studied. Out of 50 cases, majority of cases had GCS between 9 – 11 on discharge; the second largest group i.e. 14 cases (28.0%) had GCS between 12 – 14 on discharge, 2 cases had death in the study group.

Figure 13) Distribution of GCS on discharge

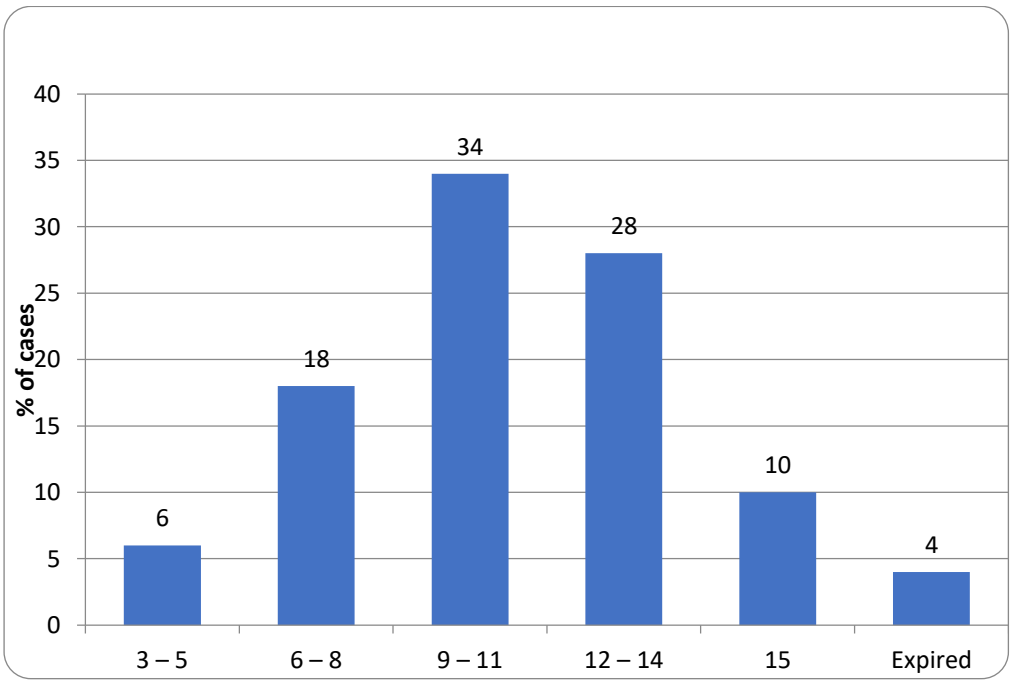


Table 14) Distribution of cases according to GOS on discharge.

GOS on discharge	No. of cases	% of cases
GOS 4 (Moderate disability)	2	4.0
GOS 3 (Severe disability)	40	80.0
GOS 2 (Vegetative state)	6	12.0
GOS 1 (Dead)	2	4.0
Total	**50**	**100.0**

Table 14 and Figure 14 shows the distribution of GOS on discharge among the cases studied. Out of 50 cases, majority of cases had GOS 3 (Severe disability) on discharge; the second largest group i.e. 6 cases (12.0%) had GOS 2 (Vegetative state), 2 cases had death in the study group.

Figure 14) Distribution of GOS on discharge

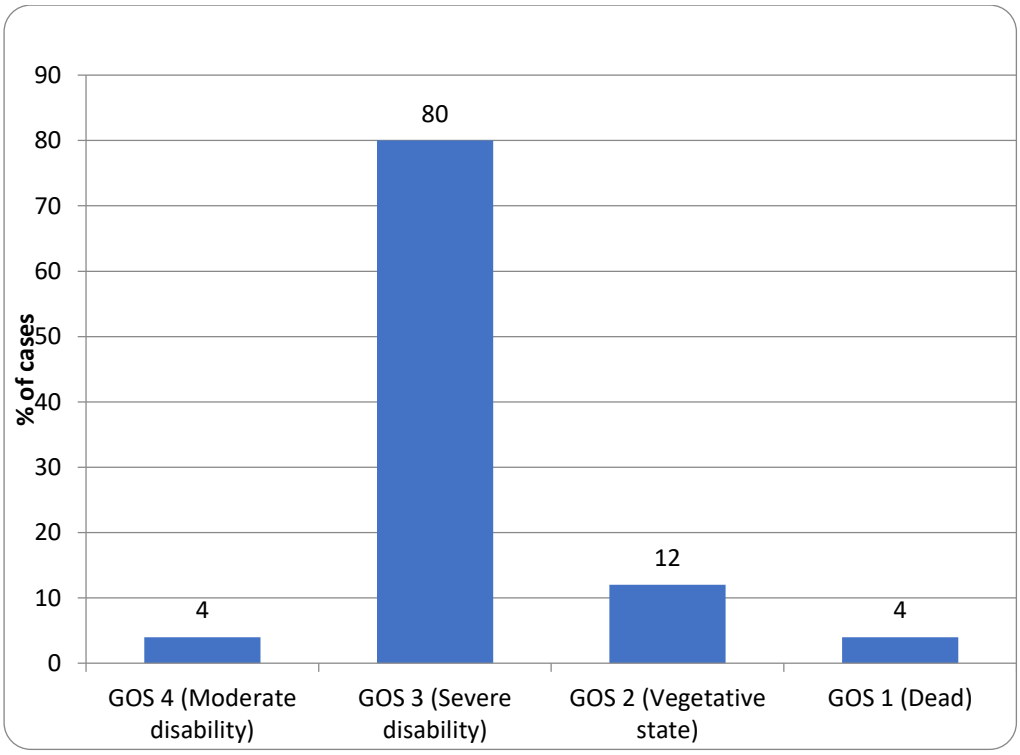


Table 15) Distribution of cases according to mRS on discharge.

Outcome	mRS Score	No. of cases	% of cases
Slight disability	2	2	4.0
Moderate disability	3	1	2.0
Moderately severe disability	4	37	74.0
Severe disability and requiring constant nursing care and attention	5	8	16.0
Dead	6	2	4.0
Total	**--**	**50**	**100.0**

Table 15 and Figure 15 shows the distribution of mRS score on discharge among the cases studied. Out of 50 cases, majority of cases (37 cases, 74%) had mRS score 4 (moderately severe disability) on discharge; the second largest group i.e. 8 cases (16.0%) had mRS score 5 (Severe disability and requiring constant nursing care and attention), 2 cases had death in the study group.

Figure 15) Distribution of mRS on discharge

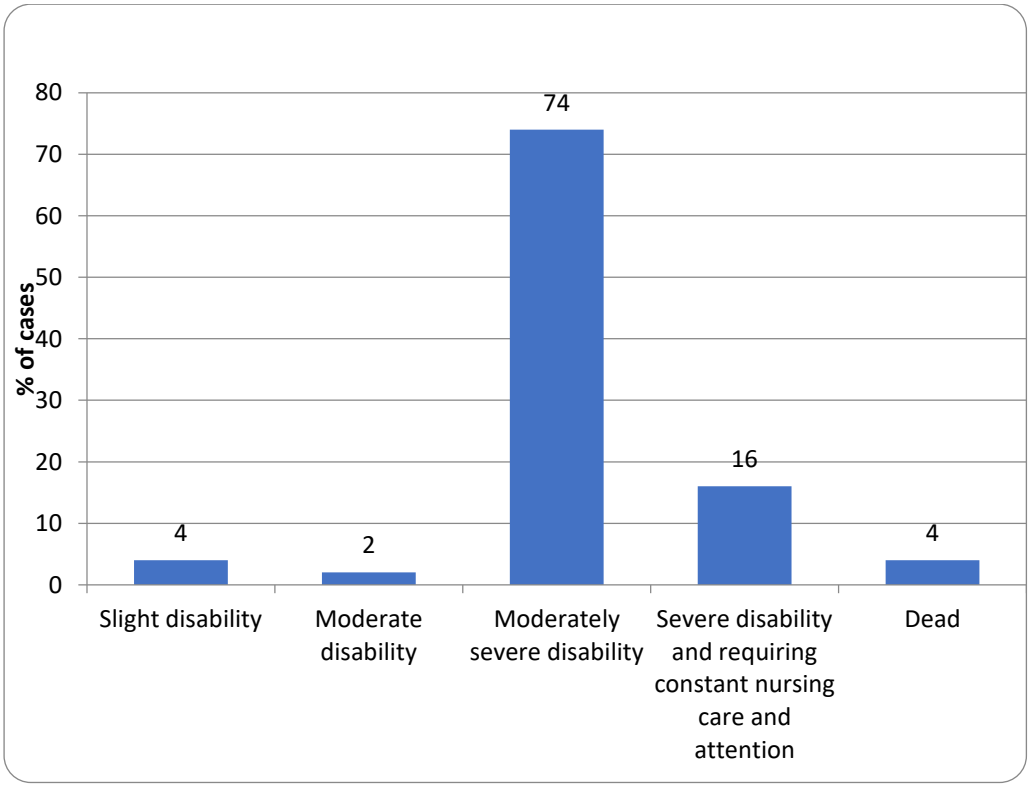


Table 16) Distribution of cases according to GCS after 2-month follow-up.

GCS after 2-month follow-up	No. of cases	% of cases
3 – 5	0	0.0
6 – 8	4	8.0
9 – 11	14	28.0
12 – 14	12	24.0
15	17	34.0
Expired	3	6.0
Total	**50**	**100.0**

Table 16 and Figure 16 shows the distribution of GCS after 2-month follow-up among the cases studied. Out of 50 cases, majority of cases had GCS between 9 – 11 after 2-month follow-up; the second largest group i.e. 17 cases (34.0%) had GCS score 15 after 2-month follow-up, 3 cases had death in the study group.

Figure 16) Distribution of GCS after 2 month follow-up

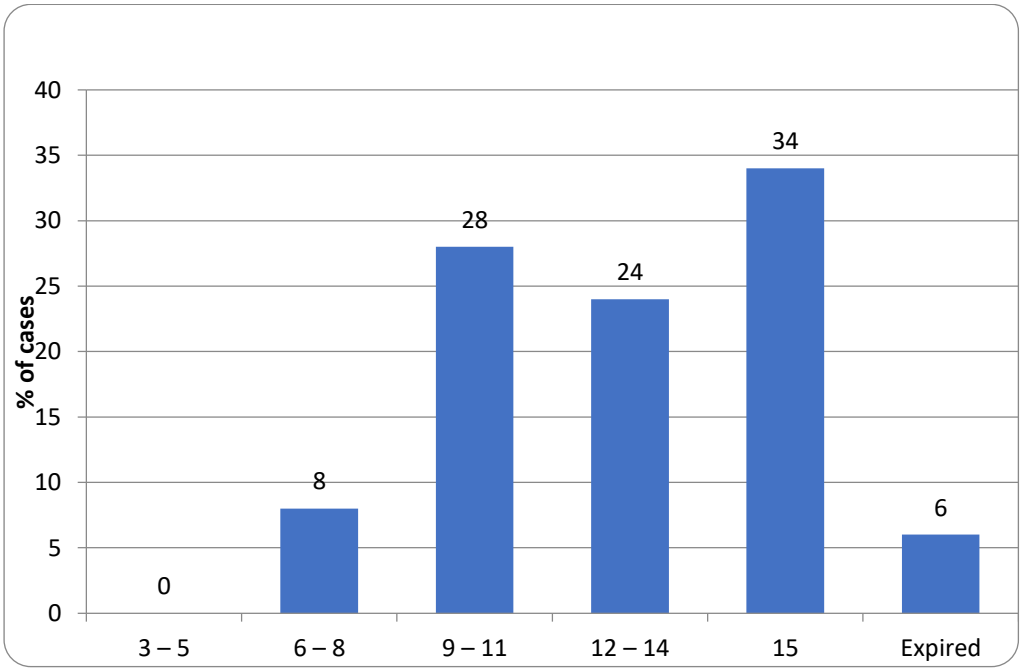


Table 17) Distribution of cases according to GOS after 2-month follow-up.

GOS after 2-month follow-up	No. of cases	% of cases
GOS 5 (Good recovery)	1	2.0
GOS 4 (Moderate disability)	14	28.0
GOS 3 (Severe disability)	31	62.0
GOS 2 (Vegetative state)	1	2.0
GOS 1 (Dead)	3	6.0
Total	**50**	**100.0**

Table 17 and Figure 17 shows the distribution of GOS after 2-month follow-up among the cases studied. Out of 50 cases, majority of cases (31 cases, 62.0%) had GOS 3 (Severe disability) after 2-month follow-up; the second largest group i.e. 14 cases (28.0%) had GOS 4 (Moderately disable), 3 cases had death in the study group.

Figure 17) Distribution of GOS after 2 month follow-up

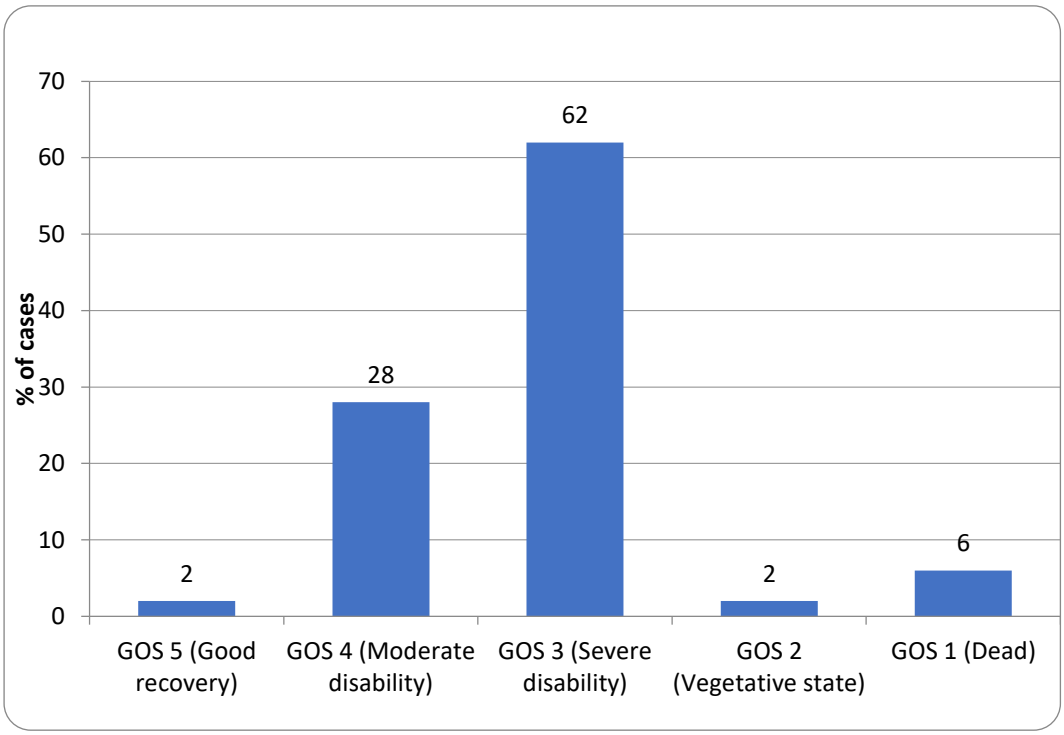


Table 18) Distribution of cases according to mRS after 2-month follow-up

Outcome	mRS Score	No. of cases	% of cases
Slight disability	2	6	12.0
Moderate disability	3	13	26.0
Moderately severe disability	4	24	48.0
Severe disability and requiring constant nursing care and attention	5	4	8.0
Dead	6	3	6.0
Total	**--**	**50**	**100.0**

Table 18 and Figure 18 shows the distribution of mRS score after 2-month follow-up among the cases studied. Out of 50 cases, majority of cases (24 cases, 48%) had mRS score 4 (moderately severe disability) after 2-month follow-up; the second largest group i.e. 13 cases (26.0%) had mRS score 3 (Moderate disability), 3 cases had death in the study group.

Figure 18) Distribution of mRS after 2 month follow-up

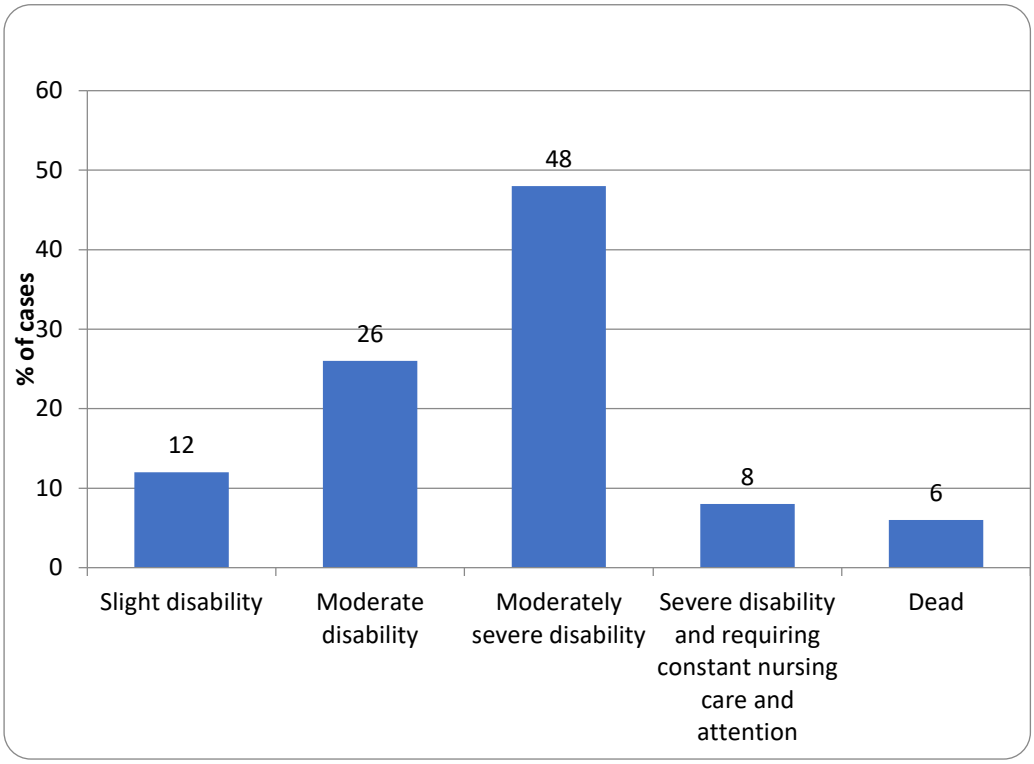


Table 19) Correlation analysis between GCS on admission and GCS on discharge and GCS after 2-month follow-up.

Correlation between	r-value	P-value
GCS on admission With GCS on discharge	0.654	0.001***
GCS on admission With GCS after 2-month	0.573	0.001***
Correlation analysis using Spearman's method. P-value<0.05 is considered to be statistically significant. ***P-value<0.001.		

Table 19 and Figure 19 shows the correlation analysis between GCS on admission with GCS on discharge and GCS after 2-month follow-up in the study group. GCS on admission showed statistically significant positive correlation with GCS on discharge and GCS after 2-month follow-up in the study group (P-value<0.05 for both).

Figure 19) Correlation analysis (GCS score)

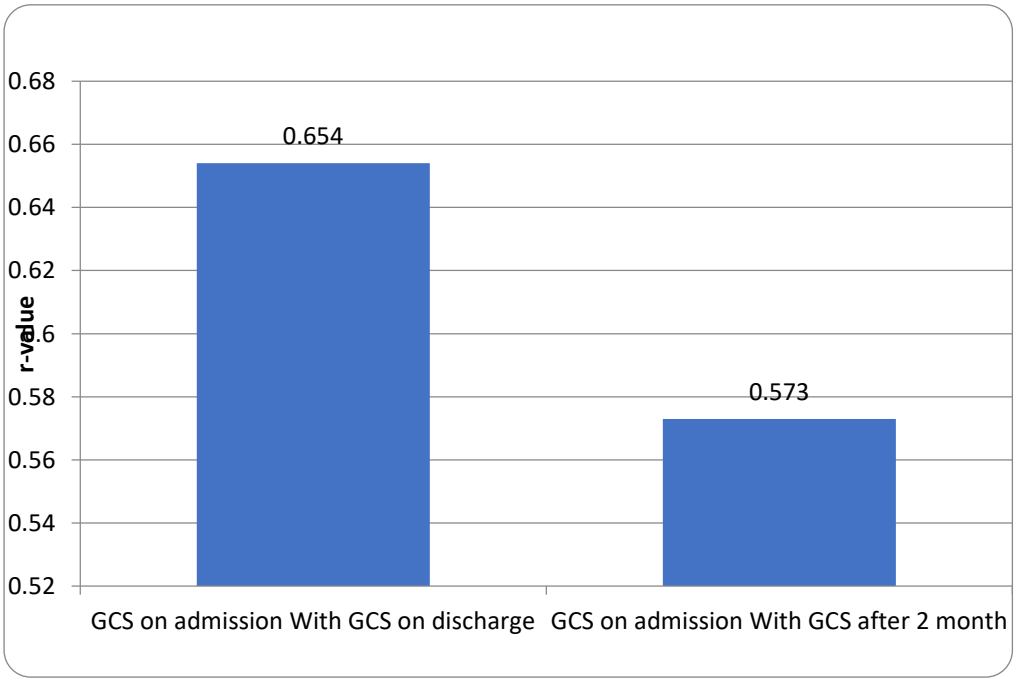


Table 20) Correlation analysis between GCS on admission and GOS on discharge and GOS after 2-month follow-up.

Correlation between	r-value	P-value
GCS on admission With GOS on discharge	0.205	0.163^{NS}
GCS on admission With GOS after 2-month	0.258	0.080^{NS}
Correlation analysis using Spearman's method. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.		

Table 20 and Figure 20 shows the correlation analysis between GCS on admission with GOS on discharge and GOS after 2-month follow-up in the study group. GCS on admission did not show statistically significant correlation with GOS on discharge and GOS after 2-month follow-up in the study group (P-value>0.05 for both).

Figure 20) Correlation analysis (GCS score With GOS score)

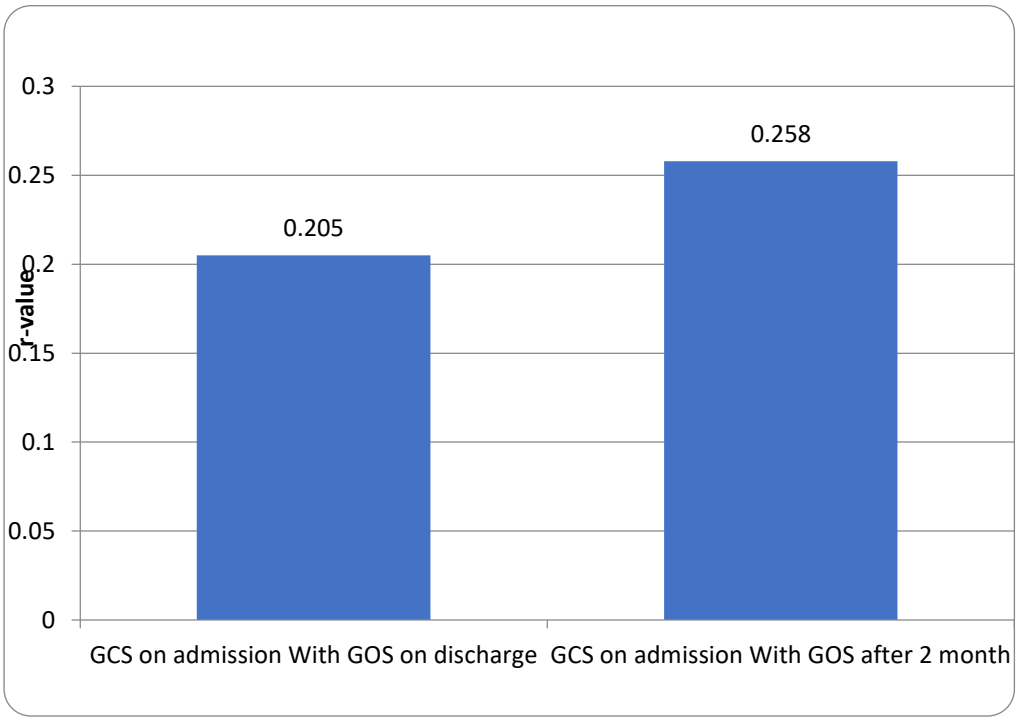


Table 21) Correlation analysis between GCS on admission and mRS on discharge and mRS after 2-month follow-up.

Correlation between	r-value	P-value
GCS on admission with mRS on discharge	-0.299	0.039*
GCS on admission with mRS after 2-month	-0.372	0.010**
Correlation analysis using Spearman's method. P-value<0.05 is considered to be statistically significant. *P-value<0.05, **P-value<0.01.		

Table 21 and Figure 21 shows the correlation analysis between GCS on admission with mRS on discharge and mRS after 2-month follow-up in the study group. GCS on admission showed statistically significant negative correlation with mRS on discharge and mRS after 2-month follow-up in the study group (P-value<0.05 for both).

Figure 21) Correlation analysis (GCS score With mRS score)

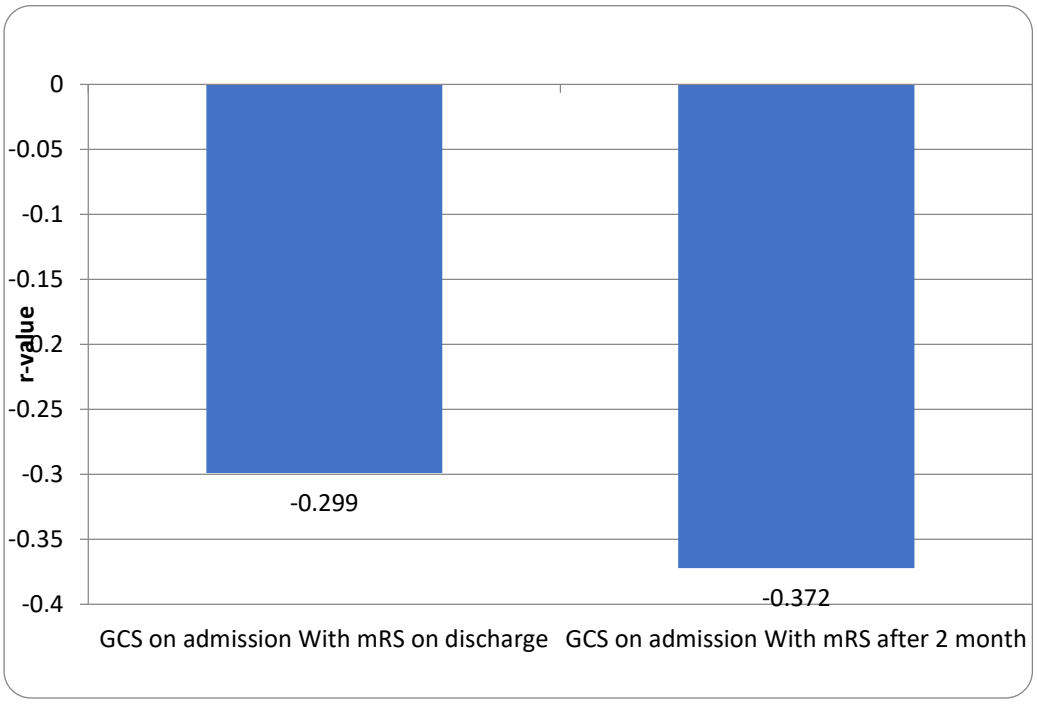


Table 22) Distribution of mean GCS according to location of haemorrhage.

		GCS Score					
		On Admission		On Discharge		After 2-month follow-up	
Location	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Let Fronto-temporal	1	8.00	--	11.00	--	13.00	--
Left Gangliocapsular	15	6.40	2.72	10.60	2.19	12.27	2.12
Left Thalamic	10/10/9 (1 pt died)	6.70	3.47	10.70	3.68	13.00	2.18
Left Parietal	1	10.00	--	11.00	--	15.00	--
Left Temporoparietal	1	11.00	--	14.00	--	15.00	--
Right Gangliocapsular	17/15/15 (2 pts died)	6.29	3.60	9.07	3.55	10.47	3.02
Right Thalamic	5	7.60	2.07	13.80	1.09	15.00	0.00
F-value (ANOVA)	--	0.644		1.810		3.098	
P-value	--	0.695 NS		0.121 NS		0.014*	

P-value by ANOVA. P-value<0.05 is considered to be statistically significant. *P-value<0.05, NS – Statistically non-significant.

Table 22 and Figure 22 shows the comparison of mean GCS according to location of haemorrhage in the study group. The distribution of mean GCS on admission and mean GCS on discharge did not differ significantly across various locations of haemorrhage in the study group (P-value>0.05 for both). Distribution of mean GCS after 2-month

follow-up differs significantly across various locations of haemorrhage in the study group (P-value<0.05). Location seems to have statistically significant impact on GCS after 2-month follow-up.

Figure 22) Distribution of mean GCS according to location of haemorrhage

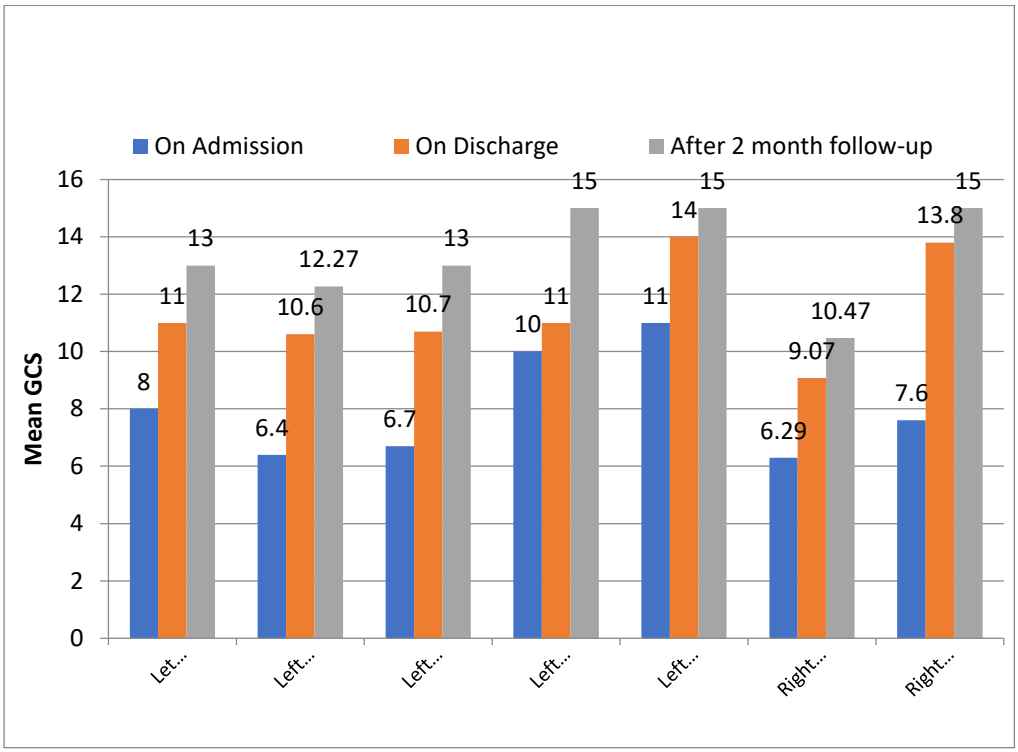


Table 23) Distribution of mean GOS according to location of haemorrhage.

		GOS Score			
		On Discharge		After 2-month follow-up	
Location	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
Let Frontotemporal	1	3.00	--	3.00	--
Left Gangliocapsular	15	3.00	0.38	3.20	0.41
Left Thalamic	10 / 9 (1 pt died)	2.90	0.32	3.56	0.53
Left Parietal	1	3.00	--	3.00	--
Left Temporoparietal	1	3.00	--	3.00	--
Right Gangliocapsular	15	2.73	0.46	3.13	0.52
Right Thalamic	5	3.20	0.45	3.80	0.84
F-value (ANOVA)	**--**	1.064		1.827	
P-value	**--**	0.399^{NS}		0.118^{NS}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 23 and Figure 23 shows the comparison of mean GOS according to location of haemorrhage in the study group. The distribution of mean GOS on discharge and GOS after 2-month follow-up did not differ significantly across various locations of haemorrhage in the study group (P-value>0.05 for both). Location seems to have no statistically significant impact on GOS on discharge and GOS after 2-month follow-up.

Figure 23) Distribution of mean GOS according to location of haemorrhage

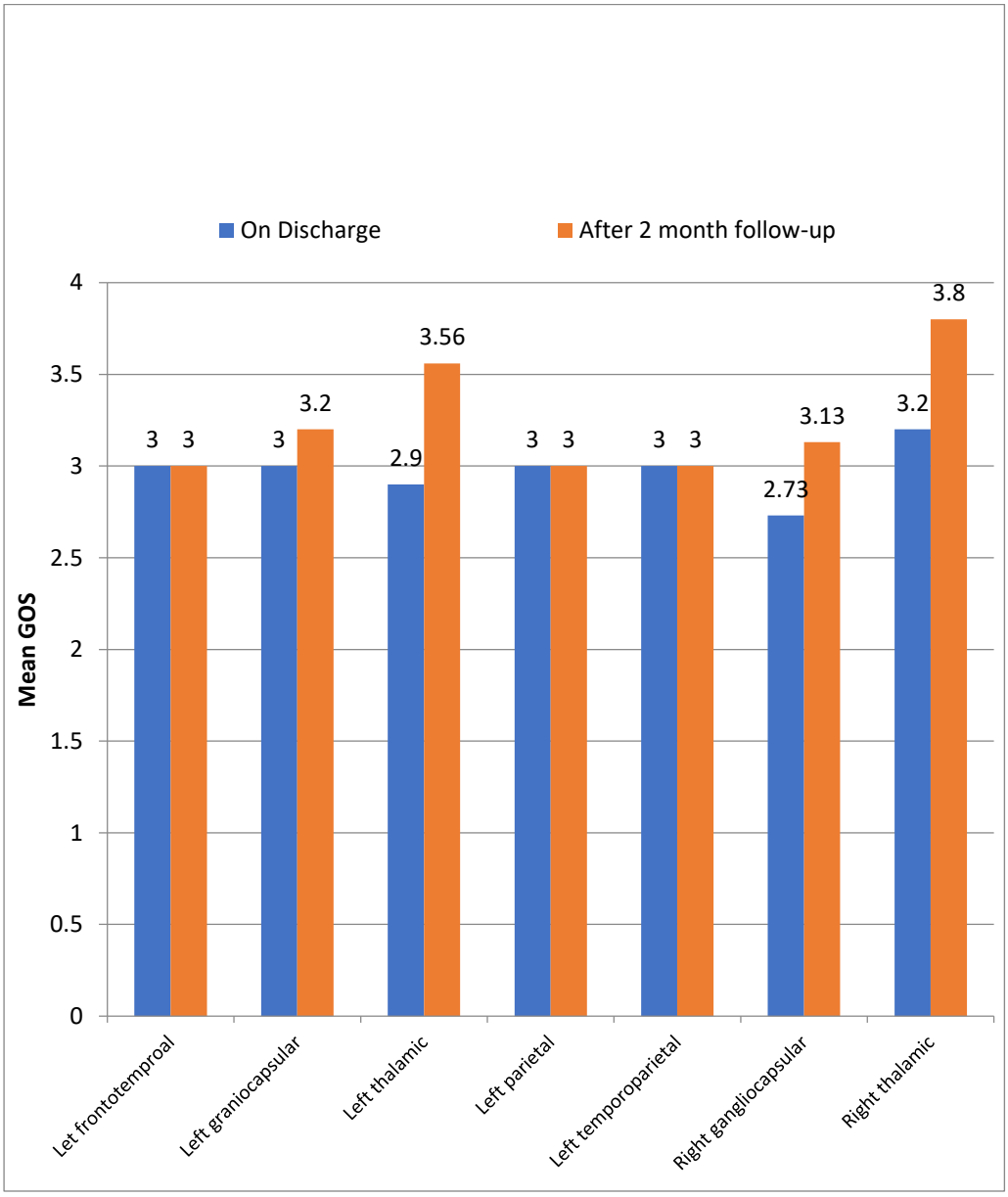


Table 24) Distribution of mean mRS according to location of haemorrhage.

		mRS Score			
		On Discharge		**After 2-month follow-up**	
Location	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
Let Frontotemporal	1	4.00	--	4.00	--
Left Gangliocapsular	15	3.93	0.88	3.73	0.88
Left Thalamic	10 / 9 (1 pt died)	4.10	0.32	3.11	0.78
Left Parietal	1	4.00	--	4.00	--
Left Temporoparietal	1	4.00	--	4.00	--
Right Gangliocapsular	15	4.27	0.46	3.80	0.77
Right Thalamic	5	3.80	0.45	3.00	0.71
F-value (ANOVA)	**--**	0.547		1.372	
P-value	**--**	0.769^{NS}		0.250^{NS}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 24 and Figure 24 shows the comparison of mean mRS according to location of haemorrhage in the study group. The distribution of mean mRS on discharge and mRS after 2-month follow-up did not differ significantly across various locations of haemorrhage in the study group (P-value>0.05 for both). Location seems to have no statistically significant impact on mRS on discharge and mRS after 2-month follow-up.

Figure 24) Distribution of mean mRS according to location of haemorrhage

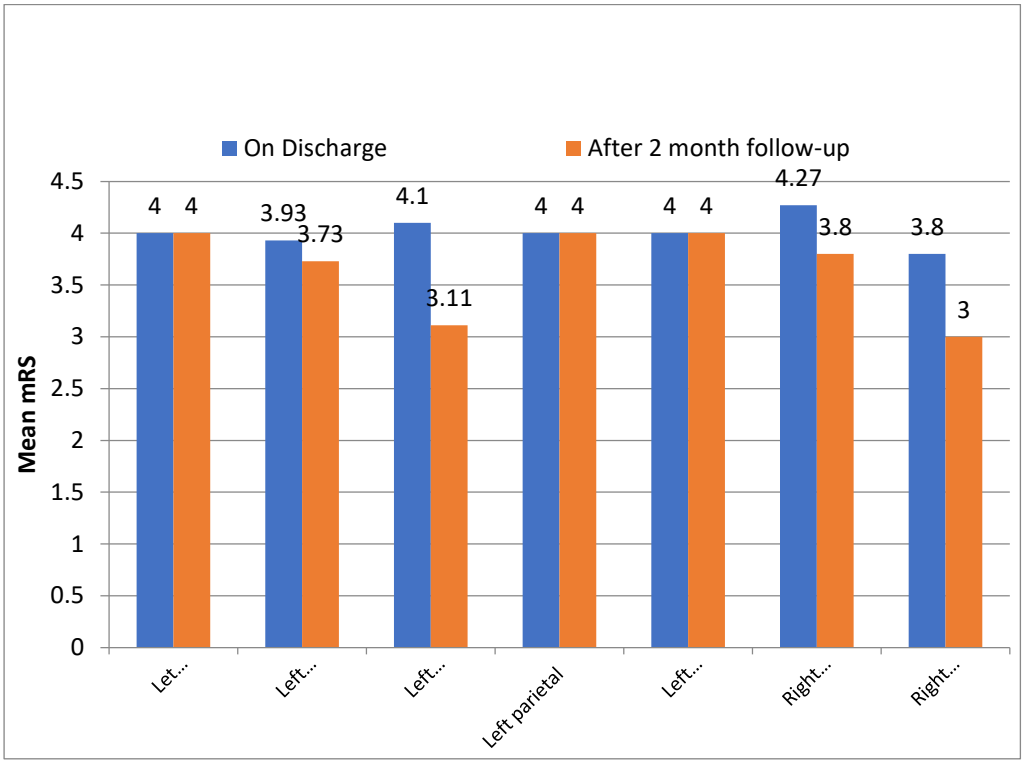


Table 25) Distribution of incidence of mortality according to location of haemorrhage.

		Mortality			
		Survived		Death	
Location	**No. of cases**	**N**	**%**	**n**	**%**
Let Frontotemporal	1	1	100.0	0	0.0
Left Gangliocapsular	15	15	100.0	0	0.0
Left Thalamic	10	9	90.0	1	10.0
Left Parietal	1	1	100.0	0	0.0
Left Temporoparietal	1	1	100.0	0	0.0
Right Gangliocapsular	17	15	88.2	2	11.8
Right Thalamic	5	5	100.0	0	0.0
Chi-Square Value	**--**	2.753			
P-value	**--**	0.839^{NS}			
P-value by Chi-Square value. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 25 and Figure 25 shows the comparison of incidence of mortality according to location of haemorrhage in the study group. The distribution of incidence of mortality did not differ significantly across various locations of haemorrhage in the study group (P-value>0.05). Location seems to have no statistically significant impact on incidence of mortality.

Figure 25) Distribution of incidence of mortality according to location of haemorrhage

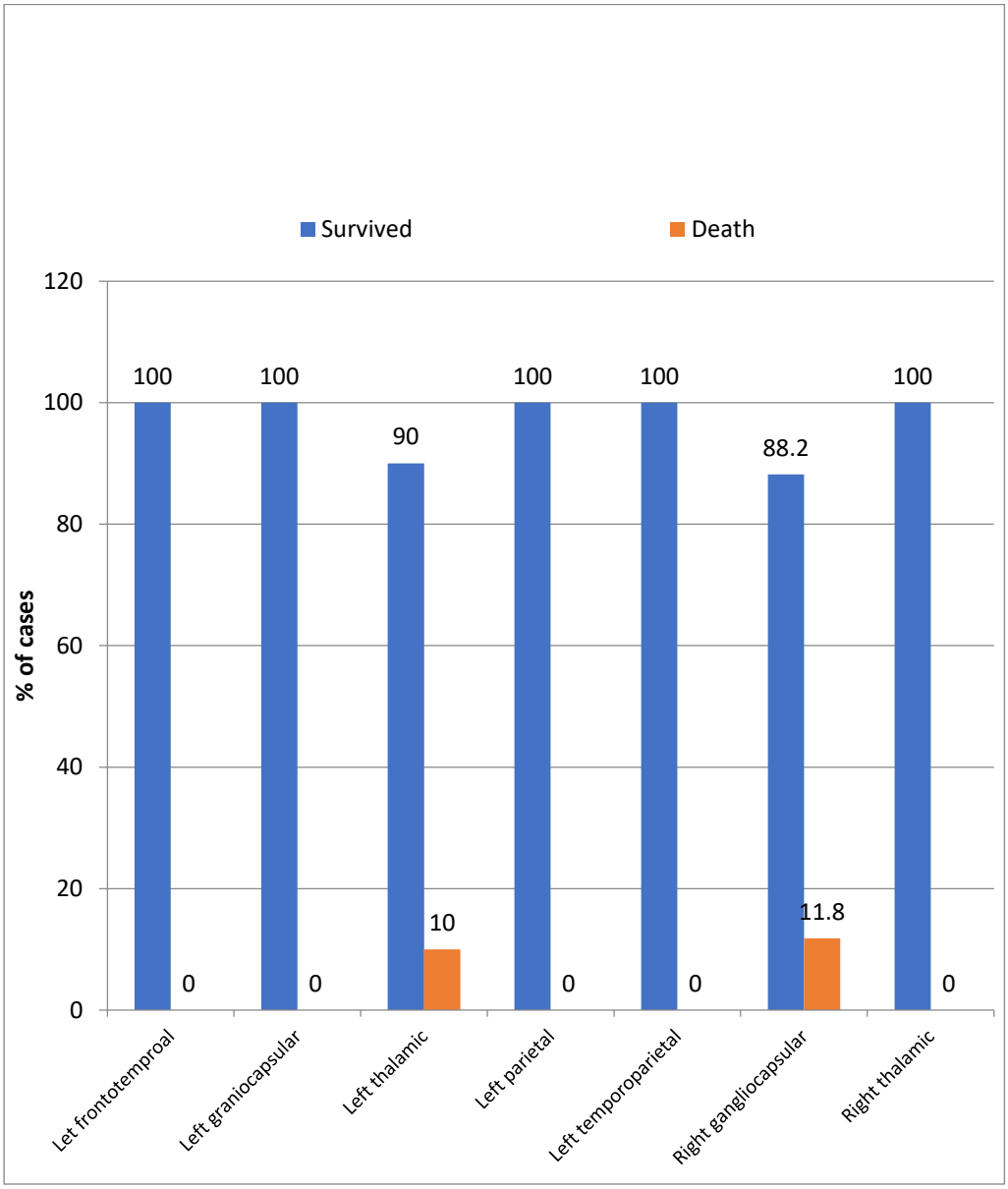


Table 26) Correlation analysis between volume of bleed and GCS, GOS and mRS.

Correlation between	r-value	P-value
Volume of bleed With GCS on admission	-0.311	0.028*
Volume of bleed With GCS on discharge	-0.498	0.001***
Volume of bleed With GCS after 2 months	-0.509	0.001***
Volume of bleed With GOS on discharge	-0.290	0.045*
Volume of bleed With GOS after 2 months	-0.365	0.012*
Volume of bleed with mRS on discharge	0.381	0.008**
Volume of bleed with mRS after 2 months	0.504	0.001***
Correlation analysis using Spearman's method. P-value<0.05 is considered to be statistically significant. *P-value<0.05, **P-value<0.01, ***P-value<0.001.		

Table 26 and Figure 26 shows the correlation analysis between volume of bleed with GCS on admission, on discharge and after 2-month follow-up, GOS and mRS on discharge and after 2-month follow-up in the study group. Volume of bleed showed statistically significant negative correlation with GCS on admission, GCS on discharge and GCS after 2-month follow-up in the study group (P-value<0.05 for all).

Volume of bleed showed statistically significant negative correlation with GOS on discharge and GOS after 2-month follow-up in the study group (P-value<0.05 for all).

Volume of bleed showed statistically significant positive correlation with mRS on discharge and mRS after 2-month follow-up in the study group (P-value<0.05 for all).

Figure 26) Correlation analysis (Volume of bleed With GCS, GOS and mRS)

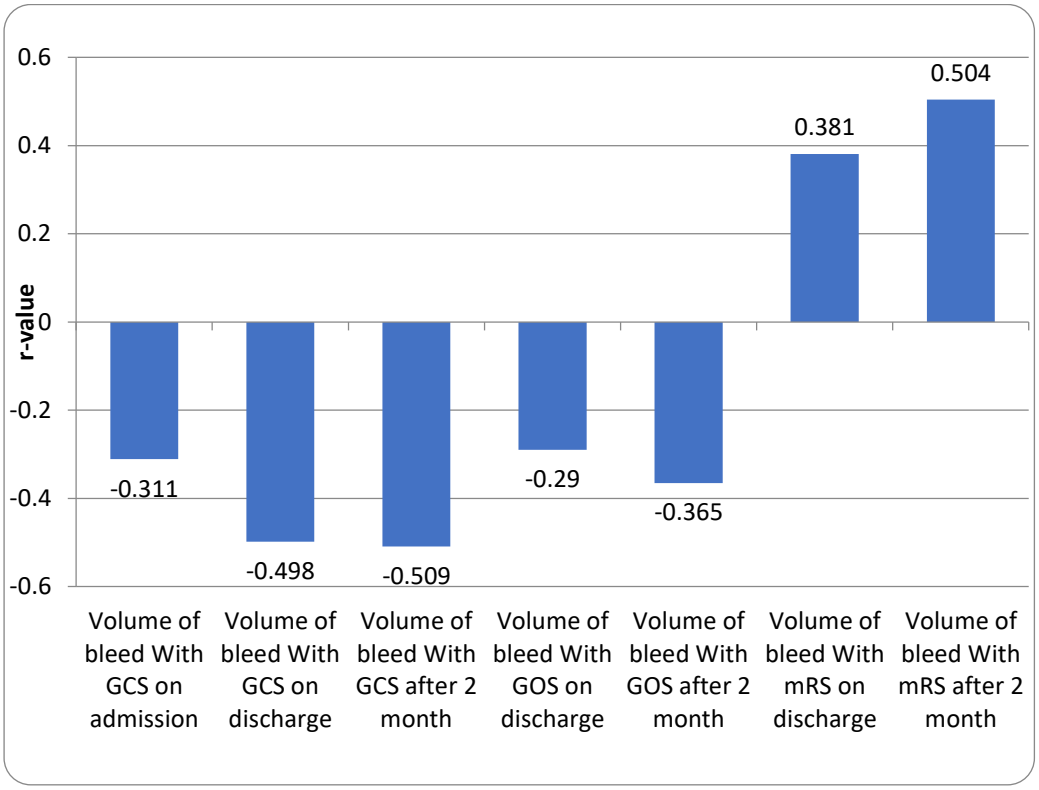


Table 27) Distribution of incidence of mortality according to volume of bleed.

		Mortality			
		Survived		Death	
Volume of bleed (ml)	No. of cases	N	%	n	%
<30	18	18	100.0	0	0.0
30 – 50	16	15	93.8	1	6.2
51 – 80	13	11	84.6	2	15.4
>80	3	3	100.0	0	0.0
Chi-Square Value	--	3.372			
P-value	--	0.338^{NS}			
P-value by Chi-Square value. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 27 and Figure 27 shows the comparison of incidence of mortality according to level of volume of bleed in the study group. The distribution of incidence of mortality did not differ significantly across various levels of volume of bleed in the study group (P-value>0.05). Volume of bleed seems to have no statistically significant impact on incidence of mortality.

Figure 27) Distribution of incidence of mortality according to volume of bleed

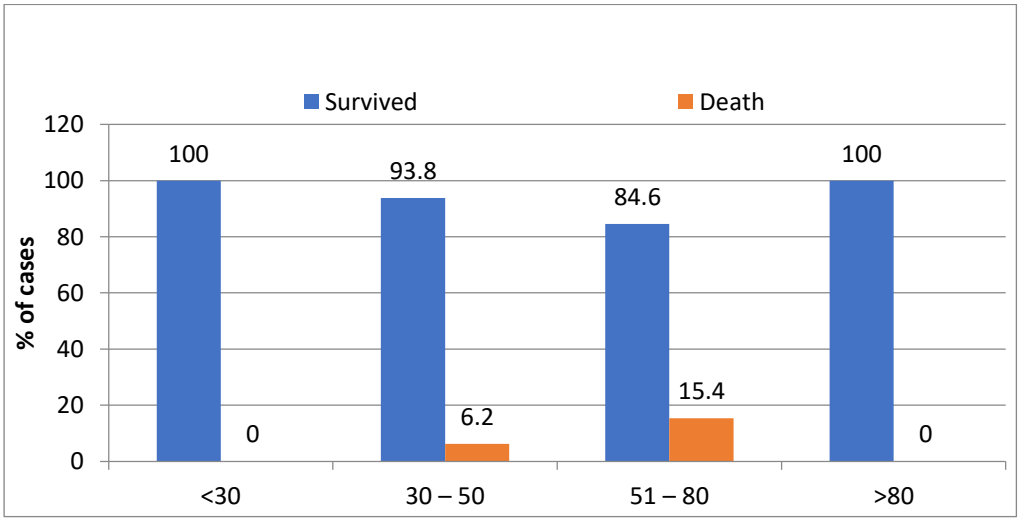


Table 28) Distribution of mean GCS according to associated IVH.

		GCS Score					
		On Admission		On Discharge		After 2-month follow-up	
Associated IVH	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Yes	35 / 33 / 32 (3 pts died)	6.03	2.98	10.18	3.19	12.00	2.59
No	15	8.40	2.85	11.40	3.02	12.80	2.93
T-value	--	2.608		1.245		0.946	
P-value	--	0.012*		0.220NS		0.349NS	
P-value by independent sample t test. P-value<0.05 is considered to be statistically significant. *P-value<0.05, NS – Statistically non-significant.							

Table 28 and Figure 28 shows the comparison of mean GCS according to associated IVH in the study group.

The distribution of mean GCS on discharge and mean GCS after 2-month follow-up did not differ significantly between group of cases with IVH and group of cases without IVH in the study group (P-value>0.05 for both).

Distribution of mean GCS on admission is significantly higher in group of cases without IVH compared to group of cases with IVH in the study group (P-value<0.05).

Figure 28) Distribution of mean GCS according to associated IVH

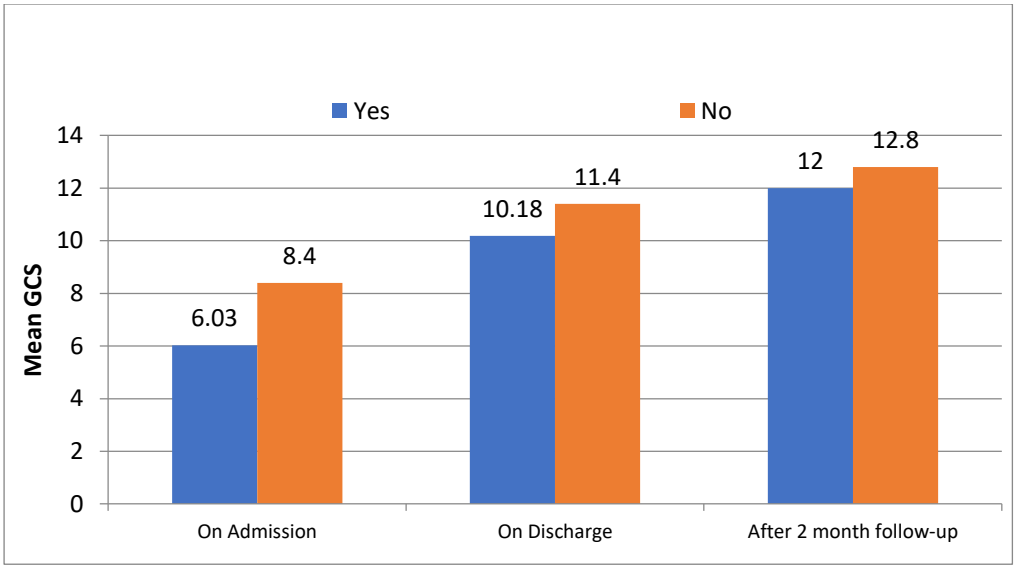


Table 29) Distribution of mean GOS according to associated IVH.

		GOS Score			
		On Discharge		After 2-month follow-up	
Associated IVH	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
Yes	33 / 32 (1 pt died)	2.94	0.43	3.41	0.56
No	15	2.87	0.35	3.13	0.52
T-value	--	0.574		1.595	
P-value	--	0.569^{NS}		0.118^{NS}	
P-value by independent sample t test. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 29 and Figure 29 shows the comparison of mean GOS according to associated IVH in the study group. The distribution of mean GOS on discharge and GOS after 2-month follow-up did not differ significantly between group of cases with IVH and group of cases without IVH in the study group (P-value>0.05 for both). Associated IVH seems to have no statistically significant impact on GOS on discharge and GOS after 2-month follow-up.

Figure 29) Distribution of mean GOS according to associated IVH

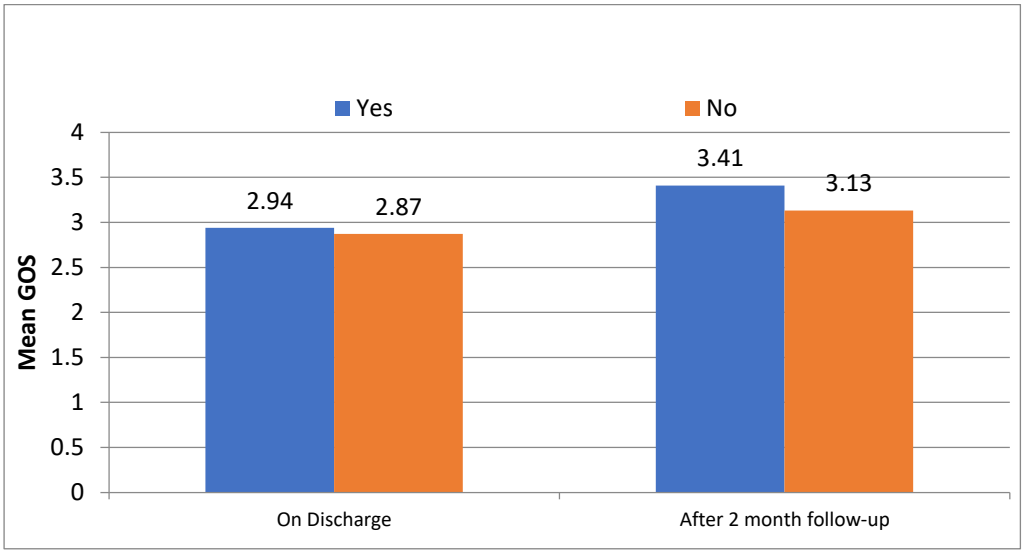


Table 30) Distribution of mean mRS according to associated IVH.

		mRS Score			
		On Discharge		After 2-month follow-up	
Associated IVH	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
Yes	33 / 32 (1 pt died)	4.03	0.68	3.50	0.92
No	15	4.13	0.35	3.67	0.62
T-value	--	0.549		0.638	
P-value	--	0.586^{NS}		0.527^{NS}	
P-value by independent sample t test. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 30 and Figure 30 shows the comparison of mean mRS according to associated IVH in the study group. The distribution of mean mRS on discharge and mRS after 2-month follow-up did not differ significantly between group of cases with IVH and group of cases without IVH in the study group (P-value>0.05 for both). Associated IVH seems to have no statistically significant impact on mRS on discharge and mRS after 2-month follow-up.

Figure 30) Distribution of mean mRS according to associated IVH

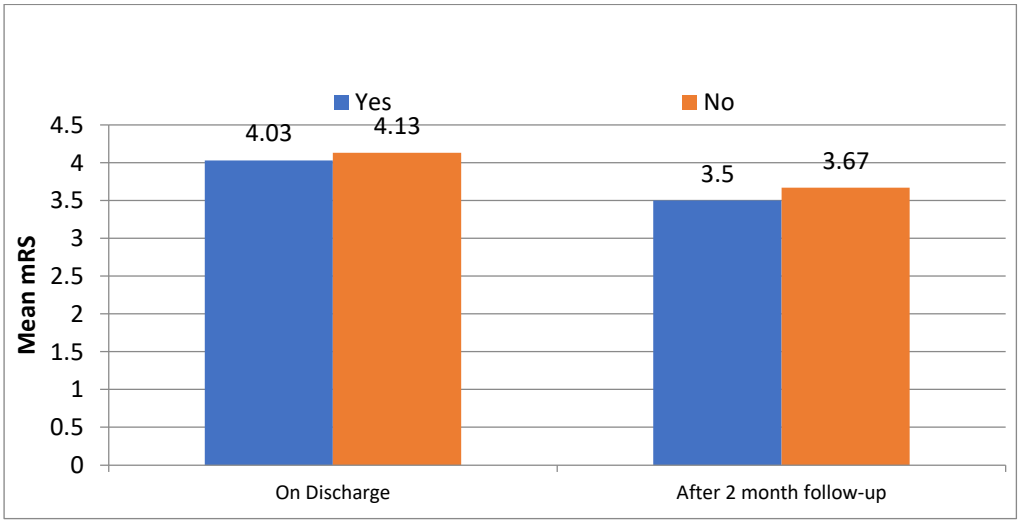


Table 31) Distribution of mean GCS according to operative technique.

		GCS Score					
		On Admission		On Discharge		After 2-month follow-up	
Operative technique	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Craniotomy and evacuation	3	8.00	3.46	11.33	2.52	13.33	2.89
DC and evacuation	28/27/27	6.64	2.96	9.78	2.95	11.33	2.70
EVD insertion	16/16/15	7.38	3.28	11.94	3.36	13.80	1.93
EVD and evacuation	3/2/2 (1 pt died)	3.00	0.00	9.00	2.83	11.50	3.54
F-value (ANOVA)	--	1.941		1.886		3.345	
P-value	--	0.136^{NS}		0.146^{NS}		0.028^{*}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. *P-value<0.05, NS – Statistically non-significant.							

Table 31 and Figure 31 shows the comparison of mean GCS according to operative technique in the study group.

The distribution of mean GCS on admission and mean GCS on discharge did not differ significantly between group of cases with various operative techniques used in the study group (P-value>0.05 for both).

Distribution of mean GCS after 2-month follow-up differs significantly across group of cases with different operative techniques used in the study group (P-value<0.05).

Figure 31) Distribution of mean GCS according to operative technique

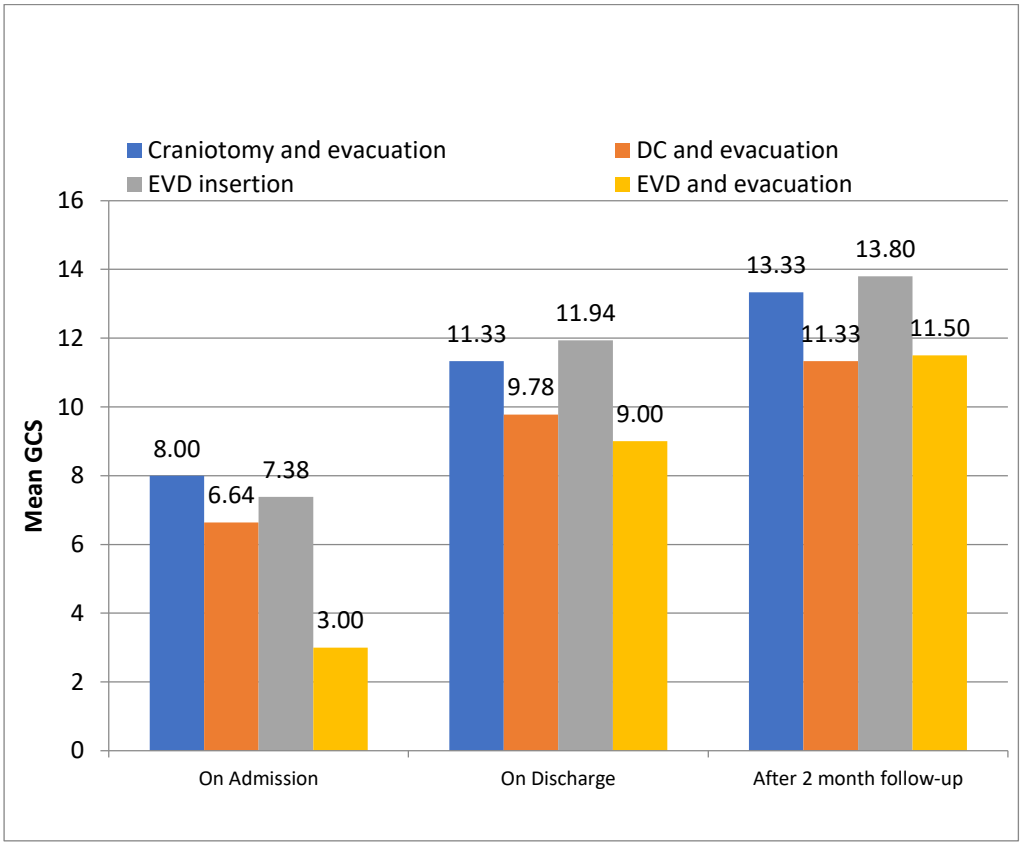


Table 32) Distribution of mean GOS according to operative technique.

		GOS Score			
		On Discharge		After 2-month follow-up	
Operative technique	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
Craniotomy and evacuation	3	3.00	0.00	3.00	0.00
DC and evacuation	27/27	2.85	0.46	3.15	0.46
EVD insertion	16/15	3.00	0.36	3.67	0.62
EVD and evacuation	2/2 (2 pts died)	3.00	0.00	3.50	0.71
F-value (ANOVA)	--	0.513		3.790	
P-value	--	0.675^{NS}		0.017^{*}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. *P-value<0.05, NS – Statistically non-significant.					

Table 32 and Figure 32 shows the comparison of mean GOS according to operative technique used in the study group. The distribution of mean GOS on discharge did not differ significantly between group of cases with various operative techniques used in the study group (P-value>0.05). The distribution of mean GOS after 2-month follow-up differs significantly between group of cases with various operative techniques used in the study group (P-value<0.05).

Operative techniques seem to have statistically significant impact on GOS after 2-month follow-up.

Figure 32) Distribution of mean GOS according to operative technique

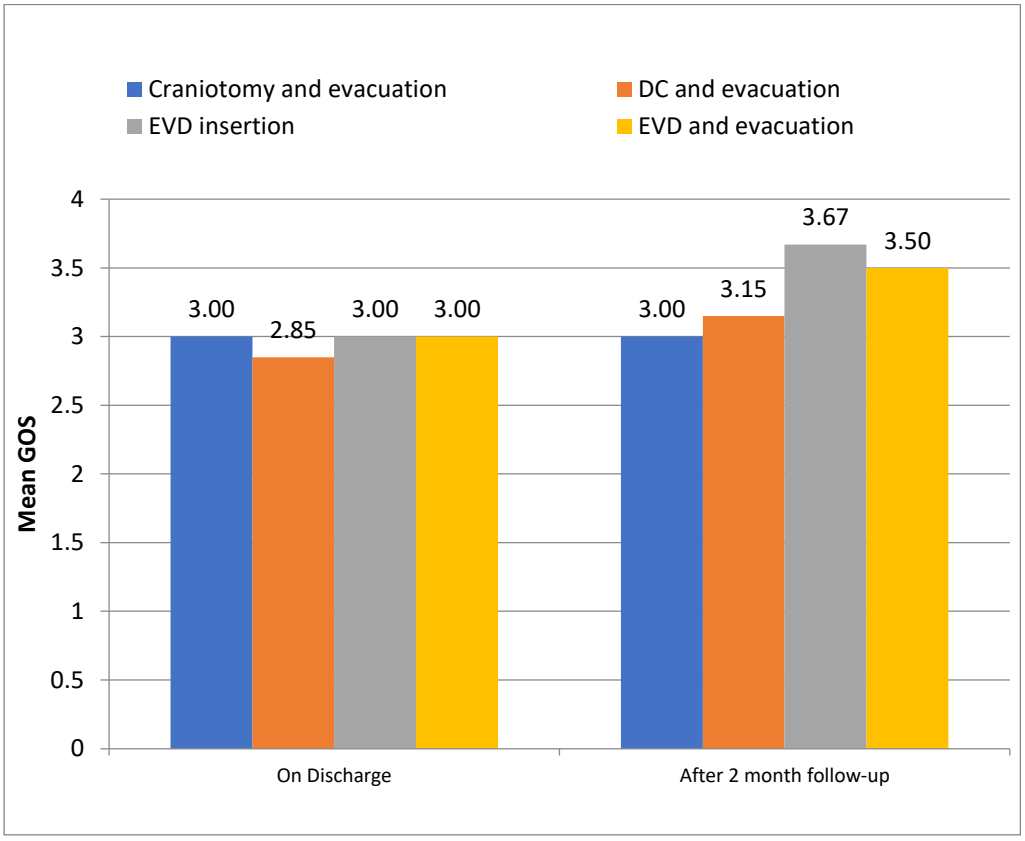


Table 33) Distribution of mean mRS according to operative technique.

		mRS Score			
		On Discharge		After 2-month follow-up	
Operative technique	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
Craniotomy and evacuation	3	4.00	0.00	4.00	0.00
DC and evacuation	27/27	4.11	0.75	3.81	0.79
EVD insertion	16/15	4.00	0.36	3.00	0.76
EVD and evacuation	2/2 (2 pts died)	4.00	0.00	3.50	0.71
F-value (ANOVA)	**--**	0.128		4.108	
P-value	**--**	0.943^{NS}		0.012*	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. *P-value<0.05, NS – Statistically non-significant.					

Table 33 and Figure 33 shows the comparison of mean mRS according to operative technique used in the study group. The distribution of mean mRS on discharge did not differ significantly between group of cases with various operative techniques used in the study group (P-value>0.05). The distribution of mean mRS after 2-month follow-up differs significantly between group of cases with various operative techniques used in the study group (P-value<0.05).

Operative techniques seem to have statistically significant impact on mRS after 2-month follow-up.

Figure 33) Distribution of mean mRS according to operative technique

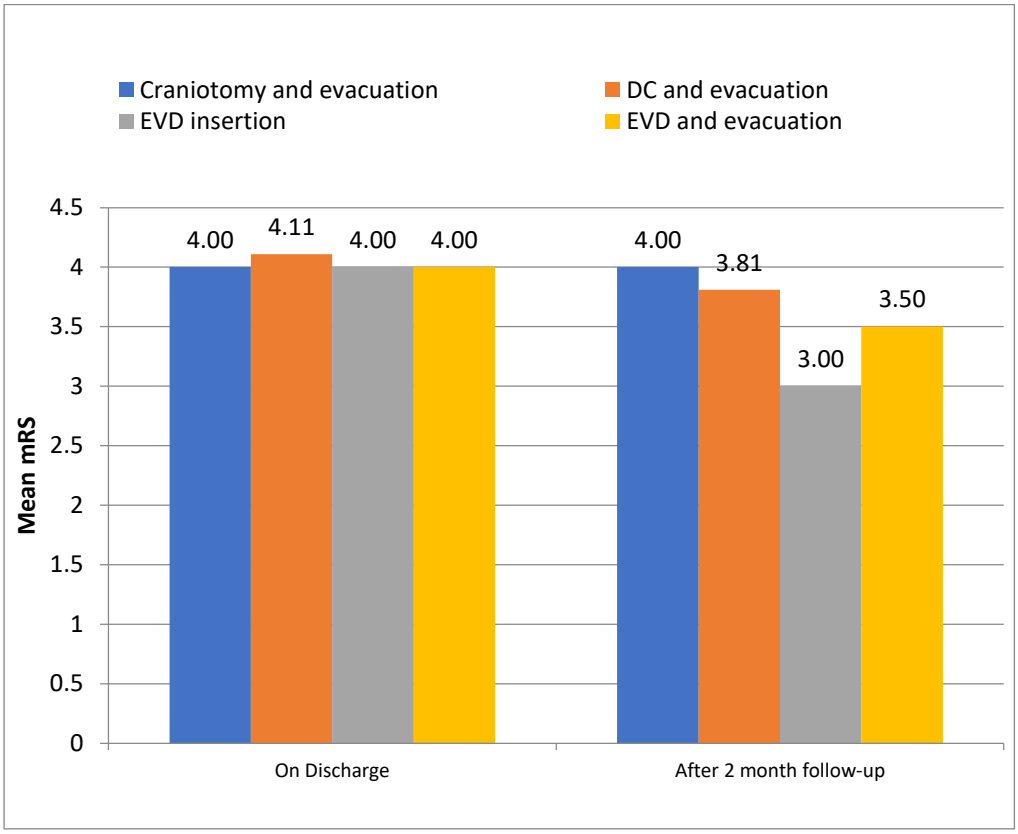


Table 34) Distribution of mean GCS according to mid-line shift.

		GCS Score					
		On Admission		**On Discharge**		**After 2-month follow-up**	
Mild-line shift	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
<5 mm	4	9.00	3.74	14.50	0.58	15.00	0.00
5 – 10 mm	34/33/32	7.00	2.97	10.64	2.91	12.44	2.47
>10 mm	12/11/11 (3 pts died)	5.25	2.89	8.91	3.24	10.73	2.97
F-value (ANOVA)	**--**	2.722		5.499		4.500	
P-value	**--**	0.076^{NS}		0.007^{**}		0.017^{*}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. *P-value<0.05, **P-value<0.01, NS – Statistically non-significant.							

Table 34 and Figure 34 shows the comparison of mean GCS according to mid-line shift in the study group.

The distribution of mean GCS on admission did not differ significantly between group of cases with various levels of mid-line shift in the study group (P-value>0.05 for both).

Distribution of mean GCS on discharge and mean GCS after 2-month follow-up differs significantly across group of cases with different levels of mid-line shift in the study group (P-value<0.05 for both).

Figure 34) Distribution of mean GCS according to mid-line shift

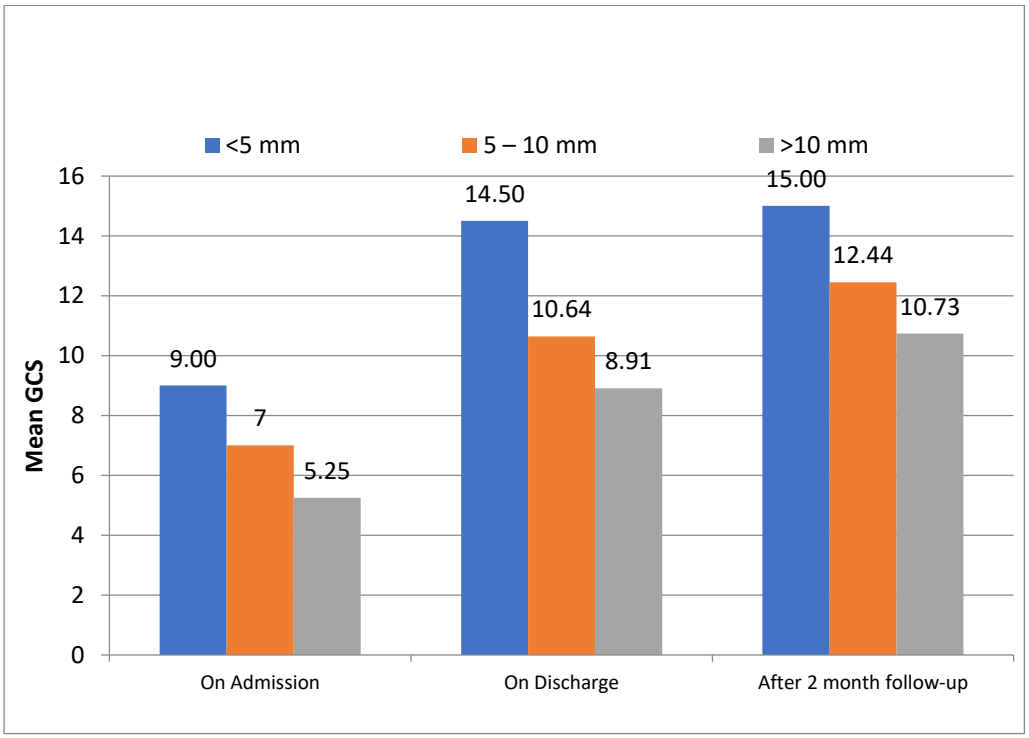


Table 35) Distribution of mean GOS according to mid-line shift.

		GOS Score			
		On Discharge		After 2-month follow-up	
Mild-line shift	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
<5 mm 5 – 10 mm >10 mm	4 33/32 11/11 (3 pts died)	3.25 2.94 2.73	0.50 0.35 0.47	3.75 3.31 3.18	0.96 0.47 0.60
F-value (ANOVA) **P-value**	**--** **--**	2.828 0.070^{NS}		1.578 0.218^{NS}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 35 and Figure 35 shows the comparison of mean GOS according to mid-line shift in the study group. The distribution of mean GOS on discharge and mean GOS after 2-month follow-up did not differ significantly between group of cases with various levels of mid-line shift in the study group (P-value>0.05 for both).

Mid-line shift seems to have no statistically significant impact on GOS on discharge and GOS after 2-month follow-up.

Figure 35) Distribution of mean GOS according to mid-line shift

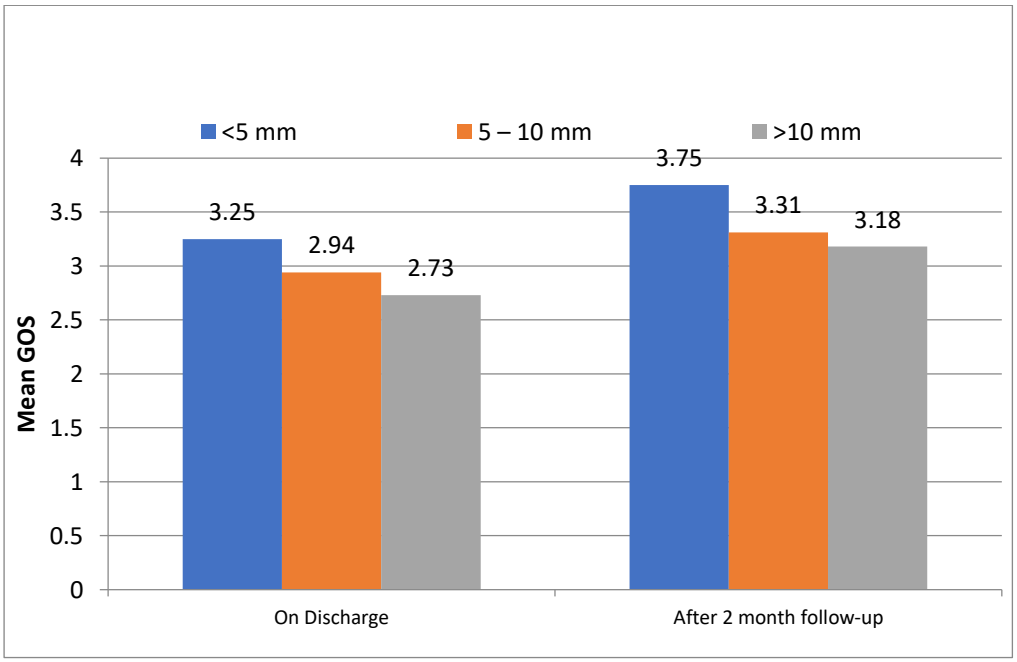


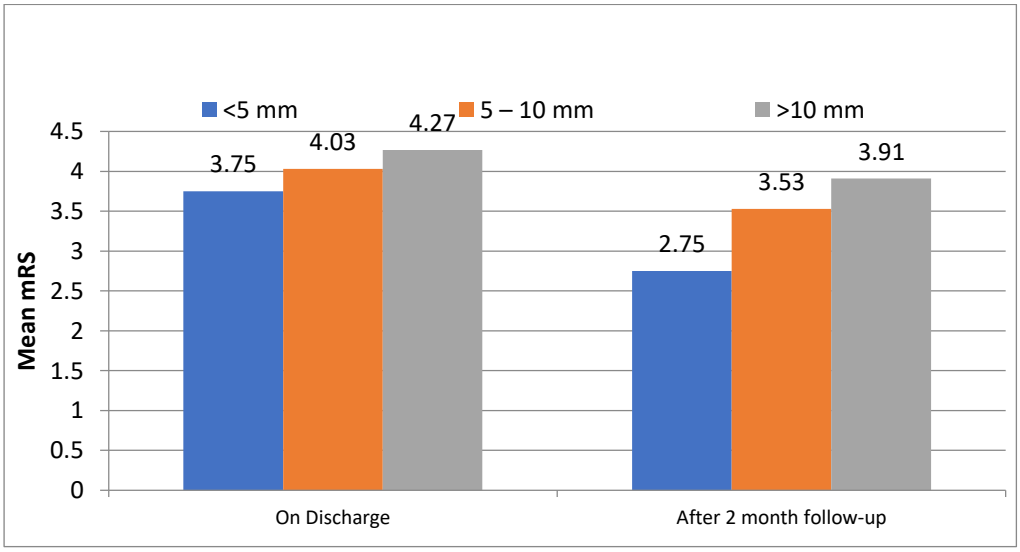
Table 36) Distribution of mean mRS according to mid-line shift.

		mRS Score			
		On Discharge		After 2-month follow-up	
Mild-line shift	**No. of cases**	**Mean**	**SD**	**Mean**	**SD**
<5 mm 5 – 10 mm >10 mm	4 33/32 11/11 (3 pts died)	3.75 4.03 4.27	0.50 0.64 0.47	2.75 3.53 3.91	0.96 0.80 0.70
F-value (ANOVA) **P-value**	-- --	1.289 0.286^{NS}		3.177 0.051^{NS}	
P-value by ANOVA. P-value<0.05 is considered to be statistically significant. NS – Statistically non-significant.					

Table 36 and Figure 36 shows the comparison of mean mRS according to mid-line shift in the study group. The distribution of mean mRS on discharge and mean mRS after 2-month follow-up did not differ significantly between group of cases with various levels of mid-line shift in the study group (P-value>0.05 for both).

Mid-line shift seems to have no statistically significant impact on mRS on discharge and mRS after 2-month follow-up.

Figure 36) Distribution of mean mRS according to mid-line shift



DISCUSSON

Although spontaneous intracerebral hemorrhage accounts for only 10-20% of strokes, it remains a devastating condition and early mortality rates are approximately 40%, with many of the survivors remaining disabled. [316] Outcome after ICH depends on the GCS score at presentation, hemorrhage size, ventricular extension, and patient's age. [317,318] There is no convincing evidence of benefit from any medical treatment. and the role of surgery remains controversial. Many clinical and radiological factors such as age, level of consciousness, HTN, Hematoma volume, volume of peripheral edema, midline shift on the initial CT scan, and intraventricular extension of hemorrhage are indicators of worse prognosis after spontaneous ICH. Very less studies have attempted to identify factors related to a favorable functional outcome in patients suffering from spontaneous supratentorial ICH. [317,319,320]

Lisk et al., observed that the GCS score, hemorrhage volume, age, and gender were important predictors of a poor outcome in patients with spontaneous ICH. [318] Daverat et al., on logistic regression analysis, found five independent predictors of a satisfactory outcome at 6 months, namely:

- Age
- Hemorrhage size
- Intraventricular spread of the hemorrhage
- Limb paresis
- CSF pathway communication disorders. [319]

In this study, we wanted to assess the ICH in a wide spectrum, including risk factors, various surgical interventions and factors affecting the outcome of the patients. In addition, the study provides data to compare with studies conducted in other populations employing similar criteria and methodology.

Age

In our study the mean age of patients was (53.58 +/- 12.92) years. Maximum patients in this study (76%) were in the age group between

41-70 years. Previous study by Muller and Radu (1981) [321] showed 2 peaks for hypertensive ICH; 51-55 years and 71.75 years and that the age was increasing now probably due to improved antihypertensive treatment.

This is similar to the findings in the study conducted by Obiako et al. in Ibadan, where patients aged 40-59 years constituted 73% of the patients with acute stroke.[322]

Caucasian studies show that ICH tends to occur in older and elderly population, with lower frequencies in younger age group. Although age is non-modifiable; this is useful prognostic indicator for patients and health care providers. On analysing the age group of patients, it is seen that the incidence after 80 years is only about 1% which probably shows the lower life expectancy of people. These factors are similar with the previous observation that advancing age increases the risk of ICH. Our study shows, there is no significant correlation between Age of patients and Mortality/GCS/GOS/mRS at admission, at discharge, at 1 month and at 2 months follow up.

Gender

Among the 50 patients enrolled, male patients constituted 42 (84%) and female patients 8 (16%). This study found male preponderance, with a male to female ratio of 5.25:1. This followed a similar trend to previously reported studies by Osuntokun et al. Desalu et al. and Sanya et al. [323-325] This male preponderance may represent a truly higher incidence of ICH in men as has been repeatedly reported worldwide. Similarly, in the study of brain herniation in ICH, conducted at SGPGIMS. Lucknow, out of 24 Patients 18 (75%) were men and only 6 (25%) were women, indicating a similar male preponderance. [326]

If the reason for the significantly higher proportion of male patients with ICH in our study is analysed it can be understood that the most important contributing factor is alcoholism prevalent among males. The overall incidence of alcoholism is 44% and among the 42 male patients, 22 had history of alcoholism. The study by Kearney et al suggested that men and women have similar overall prevalence of

hypertension all over the world. [327] Therefore, the hypertension among either sex is not the reason for increased incidence of ICH in males but it is the prevalence of alcoholism among males that makes them more susceptible to ICH. This implies that statutory measures have to be taken to curb the habit of consuming alcoholic beverages by Indians.

Comorbidities

In our study out of all 50 ICH patients, Hypertension was present in majority of the patients 38 (76%), Diabetes Mellitus in 8 (16%), coronary heart disease in 3 (6%), Chronic Kidney Disease and Hypothyroidism was present in 4(8%) patients each. It is similar to the results from the “INTERSTROKE study”, which identified self-reported history of HTN as a strongest risk factor for stroke overall. Most importantly the study found that this association was even more pronounced for intracerebral haemorrhagic stroke than for ischemic stroke. [328]

Hypertension is highly prevalent in India, and is consistently reported as a risk factor for all stroke types. Blood pressure control rates remain suboptimal, varying from 42 to 45% in patients receiving care in tertiary institutions, and non-compliance to treatment has been cited as a major reason for this trend. [329] Also, from among other risk factors like CHD, DM, CKD. Diabetes Mellitus was second common risk factor in ICH patients. Heavy alcohol intake has also been identified as another major risk factor in ICH, a conclusion that was drawn by Barbara et al in France [330] and also in the INTERSTROKE study, alcohol has a direct dose-dependent relationship with ICH. [331,332] Significant use of alcohol was present in the Patients in this study. and mainly among the men. Barbara and co-workers also found that heavy alcohol intake is associated with occurrence of stroke at younger age, Though the underlying mechanisms by which alcohol consumption increases risk of ICH remains unclear, possible effects include worsening hypertension, impaired hemostasis and decreased circulating levels of clotting factors of hepatic origin, excessive fibrinolysis and disseminated intravascular coagulation. [330]

Correlation between GCS at admission and outcome

The GCS score a standard neurological assessment tool which is reproducible and reliable. [333] Barbara et al., suggested that improved outcomes can be achieved with early surgery if the decision to operate is made within 8 hours of ictus, with hematomas of 20-50 ml, for patients with: GCS of 9 and more, or for patients aged 50-69 years. [334] In particular, they The GCS score suggested that, when the GCS is below 9, early surgery does not significantly improve outcome. This argument suggests that, once the GCS has dropped to below 9, irretrievable damage has already occurred and surgery will not be successful in rescuing the patient.

“Hossian et al.” explained that GCS <8 and volume of hematoma more than 60 cc is associated with poor outcomes after surgery. In their study death rate was 31.0% [335] In another study death occurred in 22.2% patients and all of these patients were with pre-operative GCS score 8 and were with hematoma volume >50 ml.

“Broderick et al”, also explained that pre-operative hemorrhage volume and GCS score is a stronger predictor of operative mortality in patients with ICH. [336]

“The STICH trial” (2005), excluded patients having GCS score >5 and concluded that patient with GCS score 5-8 is associated with 91.0% risk of poor outcomes. [337]

In our study there were 19 (38%) patients with GCS of 3-5, 16 (32%) with GCS of 6-8, 12 (24%) with GCS 9-11 and 3 (6%) patients with GCS of 12-15. Maximum patients were having GCS between 3 to 5. In this study there was statistically strong correlation between GCS at admission, at discharge and on follow up at 2 months.

Location of Hematoma

In the original ICH score model by Hemphill et al, the location of hematoma was classified as supratentorial and infratentorial.[338] In previous studies the distribution of ICH with supratentorial location is predominant, including in the study by Hemphill et al, where, of the 152 ICH cases that formed the cohort for the study, 80% of the ICH were supratentorial, while 20% were infratentorial. [339] In this study we

studied 50 patients of Supratentorial ICH. In our study lCH was divided into Gangliocapsular, Lobar, Thalamic Hematoma. We found that there were 32 (64%) patients were having Gangliocapsular ICH, 3 (6%) patients having Lobar ICH, 15 (30%) patients having Thalamic ICH.

Castellanos et al. observed that the cortical location of the bleeding was an independent predictor of a good short- term ICH outcome. [340] In another study, the patients with a basal ganglia hematoma had rates at 3 months (78.8%) compared to patients with a lobar hematoma (67.8%). [341] In our study clinical outcome in the form of Mortality, GCS, GOS and mRS, was best in Lobar Hematoma patients and worst in Gangliocapsular hematoma. However, the results are not statistically significant.

ICH Volume and Outcome

The volume of ICH is associated with the outcome in the ICH prediction models. [342,343] Larsen et al. observed that the acute mortality of ICH was 27%, and determinant for the immediate prognosis was consciousness level and the hematoma volume.[344] According to Mukesh K Bhaskar et al. study the mortality rate at 3 months was found to be directly proportional to the hematoma volume (P = 0.039) with 100% mortality for patients with a volume >90 ml. The mortality was 56.4% in patients with volume in the range of 31-60 ml whereas it was 81% in patients with the volume in the range of 61-90 ml. The mortality rate in the group of 31-60 ml volume was significantly lower in the surgical group (35.7%) than in the conservative group (77.8%) (P = 0.016). [341]

In our study, most of the patients were having ICH volume < 30 ml. The lowest and highest recorded volumes were < 30 ml and between 80 to 130 ml respectively. Hematoma volume between 25-30ml is associated with worse outcome. A substantial number of the patients (34.7%) and mostly those with basal ganglia hematoma also had intraventricular extension of the ICH. This is expected due to the anatomically contiguous location of the basal ganglia to the ventricles. IVE is associated with increased mortality and morbidity on account of

associated raised intracranial pressure and the risk of hydrocephalus and herniation. [345,346]

In our study, on comparison of ICH volume with GCS, GOS and mRS at different time there was negative correlation between ICH volume, GCS and GOS and positive correlation with mRS.

Mid Line Shift

Nag et al. found that the midline shift is prognostically poor only when coexisting with other factors causing a mass effect such as ventricular compression by the hematoma. [347] In another study, the death rate at 3 months was higher with an increase in the midline shift irrespective of the group; however, the death rate was significantly higher in the conservative group (90.9%) with a midline shift of more than 5 mm compared with the surgical group (63.3%), which was statistically significant (P = 0.023). [341]

In our study 4 (8%) patients had midline shift < 5mm, 34 (68%) patients had midline shift 5mm to 10mm, 12 (24%) patients had midline shit > 10mm. In our study Mid-line shift has statistically strong correlation with clinical outcome (GOS, mRS) and mortality.

Associated IVH

There is a substantial correlation between intraventricular blood and reduced consciousness at presentation. [348] Simple comparison of ICH patients with and without an IVH extension indicates that ICH with IVH significantly increases mortality. “Larsen et al.”, observed that intraventricular hemorrhage was a bad prognostic sign in the ganglionic-thalamic hematomas. [344] In another study by “Hallevi et al.”, patients with an IVH were 2 times more likely to have worse functional outcome (discharge mRS >3) as compared to patients without an IVH (P = 0.001). [349] In another study, the mortality rate was higher in patients having an intraventricular extension of the hematoma irrespective of the treatment group and was significantly higher in the Medically managed group (100%) compared to the Surgically managed group (80.6%).

In our study, there were 35 (70%) out of 50 patients having associated IVH. There is statistically significant relationship between IVH and GCS

at discharge. However, there is no statistically significant relationship between IVH and GOS, mRS on discharge and 2 month follow up and also with mortality or other clinical outcome at any time.

Operative Techniques

In previous studies, 'Decompressive craniectomy' (DC) with evacuation of hematoma was compared with evacuation of hematoma by craniotomy. "Hayes et al." in a retrospective study compared hematoma evacuation ± decompressive craniectomy. In patients with Putaminal hemorrhage, 10 patients underwent evacuation of hematoma with DC and were compared with 16 patients who underwent evacuation of hematoma by craniotomy. DC group had lower preoperative GCS (GCS <8, p=0.019). Decompressive Craniectomy in Putaminal hemorrhages shown significant improvement in midline shift and a trend toward better outcome. In patients with lobar ICH, eight patients underwent evacuation of hematoma + decompressive craniectomy and 17 patients underwent only hematoma evacuation by craniotomy. Decompressive craniectomy group had larger midline shift (p=0.022), and were right-sided hemorrhage (p=0.011). No benefit of decompressive craniectomy was found in patients with lobar hemorrhages. [350]

In our study, 28 (56%) patients underwent Decompressive Craniectomy and Evacuation of hematoma, 3 (6%) patients underwent Craniotomy and Evacuation of hematoma, 16 (32%) patients underwent External Ventricular Drainage (EVD) and 3 (6%) patients underwent EVD insertion with hematoma evacuation. In this study, maximum patients underwent Decompressive Craniectomy and Evacuation of hematoma. The major goal of surgical treatment in patients with an ICH is safe and thorough clot evacuation with maximal preservation of neurological function. Operative techniques used in our study have strong correlation with clinical outcome and mortality and among those EVD technique has more favourable clinical outcomes.

SUMMARY AND CONCLUSION

SUMMARY

This prospective study was conducted from 1st December 2022 to 30th November 2023 in the Department of Neurosurgery, Dayanand Medical College & Hospital, Ludhiana, after obtaining clearance from hospital ethics committee. This study included a prospective group of 50 patients admitted from December, 2022 to November, 2023.

The following is summary of the study:

1. Out of the 50 patients, the youngest patient in study was of 30 years and the oldest patient was of 87 years age. The average age was 53.58 years (median 52 years) with SD of 12.92 years. There was no significant correlation between age of patients and GCS/GOS/mRS at admission, at discharge, at 2 months.
2. Amongst 50 patient's 42 (84%) were male and rest 8 (16%) were female. Gender distribution showed a male preponderance.
3. Altered sensorium was presenting symptom in 47 (94%), focal Neurological Deficit in the form of paresis in 42 (84%), Vomiting in 11 (22%), Headache in 8 (16%), Seizure in 3 (6%), Loss of consciousness in 9 (18%), Aphasia in 7 (14%) and blurring of vision in 1 (2%) of patients. In present study altered sensorium, vomiting, loss of consciousness, headache were major presenting symptoms.
4. There were 19 (38%) patients with GCS of 3-5, 16 (32%) with GCS of 6-8, 12 (24%) with GCS 9-11 and 3 (6 %) patients with GCS of 12-15. Maximum patients presented with GCS between 3-5.
5. Out of 50 patients, there were 13 (26%) patients with pupillary asymmetry (anisocoria) and 37 (74%) patients without pupil asymmetry.
6. There were 38 (76%) patients who were hypertensive, 3 (6%) patients with CAD, 8 (16%) patients with DM, 4(8%) patients

with CKD, 4 (8%) patients with hypothyroidism. Most of the patients were hypertensive.

7. There were 22 (44%) out of 50 patients having history of alcoholism, 6 (12%) patients had h/o drug abuse and 4 (8%) patients had h/o smoking.
8. There were 15 (30%) patients having hemorrhage in Left Gangliocapsular region, 1 (2%) patient with hemorrhage in left fronto-temporal region,1(2%) patient having hemorrhage in Left tempo-parietal region, 10 (20%) patients having haemorrhage in left Thalamic region), 1 (2% patients having haemorrhage in left parietal region, 17 (34%) patients having hemorrhage in Right Gangliocapsular region, 5 (10%) patients had hemorrhage in Right thalamic region. Maximum patients were having hemorrhage in Right Gangliocapsular region.
9. There were 18 (36%) patients having ICH volume less than 30 ml, 16 (32%) patients having ICH volume 30-50 ml, 13 (26%) patients having ICH volume between 51- 80 ml and 3 (6%) patients having ICH volume more than 80 ml. Maximum patients were having ICH volume less than 30 ml.
10. 4 (8%) patients had midline shift < 5mm, 34 (68%) patients had midline shift 5mm to 10mm, 12 (24%) patients had midline shift > 10mm.
11. There were 35 (70%) patients having associated IVH.
12. 3 (6%) underwent Craniotomy and Hematoma evacuation, 28 (56%) underwent Decompressive Craniectomy and Hematoma evacuation and 16 (32%) underwent External Ventricular Drainage (EVD) and 3 (6%) patients undergone EVD with hematoma evacuation.
13. At the time of discharge 3 (6%) patient had GCS score 3 to 5, 9 (18%) patients had GCs score 6 to 8, 17 (34%) patients had GGS Score 9 to 11, 14 (28%) patients had GCS score 12 to 14, 5 (10%) patients had GCS score 15 while 2 (4%) patients expired.

14. At the time of discharge 2 (4%) patients had GOS score of 4 showing moderate disability, 40 (80%) patients had GOS score 3 showing severe disability, 6 (12%) showed GOS score 2 vegetative state while 2 (4%) patients had GOS score 1 meaning Dead.
15. At the time of discharge 2 (4%) patient had slight disability with mRS score 2, 1 (2%) had moderate disability with score 3, 37 (74%) patients had moderately severe disability with score 4, 8 (16%) patients had severe disability with score 5 while 2 (4%) patients died.
16. After 2 months 4 (8%) patients had GCS score between 6 to 8, 14(28%) had score between 9 to 11, 12 (24%) had score between 12 to 14, 17 (34%) had score 15 while 3 (6%) patients expired.
17. After 2 months 1 (2%) patient had GOS score 5 showing Good Recovery, 14 (28%) had score of 4 showing moderate disability, 31 (62%) had score 3 showing severe disability, 1 (2%) had score 2 showing vegetative state while 3 (6%) patients died.
18. After 2 months, 6 (12%) with score 2 showing slight disability, 13 (26%) having score 3 showed moderate disability, 24 (48%) had score 4 Showing moderately severe disability, 4 (8%) had score 5 showing Severe disability while 3 (6%) patients died.
19. There was statistically strong correlation between GCS at admission to GCS/GOS and mRS at discharge and on follow up after 2 months (p value <0.05). Patients with low GCS (< 8) were having poor clinical outcome and more mortality.
20. There was statistically strong correlation between GOS/mRS at discharge and on follow up after 2 months (p value<0.05)
21. To compare the GCS score on admission, at discharge, after 2 months with different location of hematoma, we found that location seems to have statistically significant impact on GCS after 2 months follow up.
22. On comparing the GOS and mRS score at discharge, after 2 months with different location of hematoma, we found that there

is some association but no statistically significant relationship. So, we conclude that the location of hematoma has no significant impact on clinical outcome.

23. On comparison of GCS, GOS and mRS at different time with ICH volume, there was statistically strong correlation found. Therefore, it concludes that ICH volume has significant impact on clinical outcome.
24. There is statistically strong correlation between IVH and GCS on discharge. However, there is no statistical correlation between IVH and GCS at admission and 2 months.
25. There is some association but not statistically significant correlation between IVH and GOS/ mRS at discharge and 2 months ($p>0.05$)
26. There exists a statistically significant variations between operative techniques and GCS at discharge and subsequent follow ups. EVD technique have more favourable outcomes on GCS at discharge (Mean ± SD 11.94+3.36) and 2 months follow-up (Mean ± SD 13.80+1.93).
27. There exists a statistically significant variations among operative techniques and GOS at discharge. EVD technique have more favourable outcomes on GOS at discharge (Mean ± SD 3.00+0.36) and 2 month (Mean + SD 3.67+0.62).
28. There exists a statistically significant variations among operative techniques and mRS at discharge. DC and evacuation technique have more favourable outcomes on mRS at discharge (Mean t SD 4.11+0.75) and 2 month (Mean ± SD 3.81+0.79).
29. The mid-line shift has a significant impact on GCS at discharge. However, at 2 month the mid-line shift has association but no Statistically significant correlation with GCS outcome. It concludes that mid-line shift affects GCS at the time of discharge and 2 months follow up.
30. The midline shift has no statistically significant impact on GOS and mRS at discharge and after 2 months.

CONCLUSION

In conclusion, an elderly male with associated comorbidities mainly Hypertension, Diabetes mellitus and Chronic alcoholic patients are at more risk to develop ICH. Clinical presentations including low GCS, Neurological deficit, Headache and Loss of consciousness have poor clinical outcome. The most common location for ICH is Gangliocapsular region followed by Thalamic region; however, the specific location of the hematoma doesn't influence overall clinical outcome or mortality rate. Maximum patients have hematoma volume less than 30 ml in the study and ICH volume plays a key role in clinical outcome of the patient. Midline shift has impact on clinical outcome and mortality i.e. patients with more midline shift has poor clinical outcome. Various operative techniques employed in managing ICH have shown significant effects on clinical outcome and mortality.

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KEY TO MASTERCHART

CAD: Coronary artery disease

CKD: Chronic kidney disease

DC: Decompressive craniectomy

DOA: Date of admission

DOS: Date of surgery

DM: Diabetes mellitus

EVD: External ventricular drain

F: Female

GC: Gangliocapsular

GCS: Glassgow coma scale

GOS: Glassgow outcome scale

HTN: Hypertension

HCP: Hydrocephalus

IVH: Intraventricular hemorrhage

LT: Left

LOC: Loss of consciousness

M: Male

mRS: Modified Rankin scale

MS: Midline shift

NAD: No abnormality detected

RT: Right

TP: Tempero-Parietal

SR NO	**MRD NO**	**AGE**	**SEX**	**OCCUPATION**	**DIAGNOSIS**	**DOA**	**DOS**	**PRESENTATION**						
								HEADACHE	VOMITTING	SEIZURES	FOCAL DEFICIT	LOC	ALTERED SENSORIUM	OTHERS
1	1539938	34 Y	M	FARMER	RT GC BLEED	21/06/23	21/06/23	-	-	-	LT HEMIPARESIS	-	YES	-
2	1540539	79 Y	M	RETIRED	LT THALAMIC BLEED	23/06/23	23/06/23	-	YES	-	-	YES	YES	APHASIA
3	1521287	40 Y	M	BARBER	LT GC BLEED	22/05/23	22/05/23	-	-	-	RT HEMIPARESIS	-	YES	-
4	1531645	40 y	M	FARMER	LT THALAMIC BLEED	07/06/23	07/06/23	-	-	-	RT HEMIPARESIS	-	YES	-
5	1527435	65 Y	M	RETIRED	RT GC BLEED	01/06/23	01/06/23	-	-	-	LT HEMIPARESIS	-	YES	-
6	1511179	45 Y	M	BUSINESSMAN	LT THALAMIC BLEED	05/05/23	05/05/23	YES	-	-	-	-	YES	-
7	1442840	67 Y	M	FARMER	LT GC BLEED	06/01/23	06/01/23	-	-	-	RT HEMIPARESIS	-	YES	-
8	1437779	45 Y	M	PVT JOB	LT FT ICH	26/12/22	26/12/22	-	-	-	RT HEMIPARESIS	YES	YES	-
9	374559	87 Y	M	RETIRED	RT GC BLEED	27/12/22	27/12/22	-	-	-	LT HEMIPARESIS	YES	YES	-
10	1439363	52 Y	M	PVT JOB	LT GC BLEED	30/12/22	30/12/22	-	YES	-	RT HEMIPARESIS	-	YES	APHASIA
11	1466777	35 Y	M	NURSING ASSIST	RT GC BLEED	17/01/23	17/01/23	-	-	-	-	-	YES	-
12	1453248	44 Y	F	HOUSEWIFE	RT GC BLEED	29/01/23	29/01/23	-	-	-	LT HEMIPARESIS	YES	YES	-
13	1439834	50 Y	F	HOUSEWIFE	LT GC BLEED	30/12/22	30/12/22	-	-	-	RT HEMIPARESIS	-	YES	-
14	1442840	67 Y	M	FARMER	LT GC BLEED	06/01/23	06/01/23	-	-	-	RT HEMIPARESIS	-	YES	-
15	1457358	54 Y	M	TEACHER	LT THALAMIC BLEED	05/02/23	05/02/23	YES	-	-	-	-	-	-
16	1449197	61 Y	M	RETIRED	LT GC BLEED	21/01/23	21/01/23	-	YES	-	RT HEMIPARESIS	-	YES	-
17	1442840	67 Y	M	FARMER	LT GC BLEED	06/01/23	06/01/23	-	-	-	RT HEMIPARESIS	-	YES	-
18	1471955	53 Y	M	FARMER	RT THALAMIC BLEED	28/02/23	28/02/23	-	-	-	LT HEMIPARESIS	-	YES	-
19	1475987	42 Y	M	SHOP KEEPER	LT GC BLEED	06/03/23	09/03/23	-	-	-	RT HEMIPARESIS	-	YES	-
20	1487818	45 Y	M	FARMER	LT THALAMIC BLEED	25/03/23	25/03/23	YES	-	-	-	-	YES	APHASIA
21	1464687	61 Y	M	FARMER	RT THALAMIC BLEED	16/02/23	17/02/23	-	-	-	LT HEMIPARESIS	-	YES	-
22	1463333	60 Y	M	FARMER	RT GC BLEED	14/02/23	14/02/23	-	-	-	LT HEMIPARESIS	-	YES	-
23	1459564	38 Y	M	LABOURER	RT GC BLEED	08/02/23	11/02/23	-	-	-	LT HEMIPARESIS	-	YES	-
24	1461871	60Y	M	RETIRED	RT GC BLEED	12/02/23	12/02/23	-	-	YES	LT HEMIPARESIS	-	YES	-
25	1476110	65 Y	F	HOUSEWIFE	LT THALAMIC BLEED	07/03/23	07/03/23	YES	-	-	RT HEMIPARESIS	-	YES	-
26	1488696	41 Y	M	GOVT SERVICE	LT GC BLEED	28/03/23	28/03/23	-	-	-	RT HEMIPARESIS	-	YES	-
27	1491189	65 Y	M	PVT JOB	LT GC BLEED	01/04/23	01/04/23	-	-	-	RT HEMIPARESIS	-	YES	-
28	778714	52 Y	M	PVT JOB	RT GC BLEED	02/04/23	14/04/23	-	-	-	LT HEMIPARESIS	-	YES	-
29	1465108	65 Y	M	PVT JOB	RT GC BLEED	13/04/23	17/04/23	-	-	-	LT HEMIPARESIS	-	YES	-
30	1502106	55 Y	M	GOVT SERVICE	LT GC BLEED	20/04/23	20/04/23	-	-	-	RT HEMIPARESIS	-	YES	APHASIA
31	1505594	60 Y	M	FARMER	LT THALAMIC BLEED	25/04/23	25/04/23	-	-	-	-	-	YES	-
32	1502106	55 Y	M	GOVT SERVICE	LT GC BLEED	20/04/23	20/04/23	-	-	-	RT HEMIPARESIS	-	YES	APHASIA
33	1553678	83Y	M	FARMER	RT GC BLEED	17/07/23	17/07/23	-	-	-	LT HEMIPARESIS	YES	YES	-
34	1546909	58 Y	M	BUSINESSMAN	LT GC BLEED	04/07/23	04/07/23	-	-	-	RT HEMIPARESIS	YES	YES	-
35	1552656	35 Y	M	GOVT SERVICE	RT GC BLEED	14/07/23	14/07/23	-	-	-	LT HEMIPARESIS	YES	YES	-
36	1572990	40 Y	M	LABOURER	RT THALAMIC BLEED	18/08/23	18/08/23	-	YES	-	LT HEMIPARESIS	-	YES	-
37	1558327	44 Y	M	TEACHER	LT TP ICH	24/07/23	25/07/23	-	-	-	RT HEMIPARESIS	-	YES	BLURRING OF VISION
38	1560440	75 Y	M	FARMER	RT THALAMIC BLEED	28/07/23	28/07/23	-	-	-	-	-	YES	-
39	1537330	30 Y	M	PVT JOB	LT THALAMIC BLEED	17/06/23	17/06/23	-	-	-	RT HEMIPARESIS	-	YES	-

SR NO	**MRD NO**	**AGE**	**COMORBIDITY**	**ADDICTION**				**SYSTEMIC EXAMINATION**	**NEUROLOGICAL EXAMINATION**		**INVESTIGATION (RADIOLOGICAL PARAMETERS**
			-	ALCOHOL	SMOKING	NSAID INTAKE	DRUG ABUSE	-	GCS	PUPILLARY ASYMMETRY	LOCATION
1	1539938	34 Y	DM, HTN	-	-	-	-	NAD	E1VetM2=3	YES	RT GC
2	1540539	79 Y	HTN	-	-	-	-	NAD	E1VetM2=3	NO	LT THALAMIC
3	1521287	40 Y	HTN	YES	-	-	-	NAD	E1VetM2=3	YES	LT GC
4	1531645	40 y	HTN	YES	-	-	-	NAD	E3V1M4=8	NO	LT THALAMIC
5	1527435	65 Y	HTN	-	-	-	-	NAD	E1VetM2=3	NO	RT GC
6	1511179	45 Y	HTN	-	-	-	-	NAD	E1VetM2=3	NO	LT THALAMIC
7	1442840	67 Y	HTN	-	-	-	-	NAD	E1V2M3=6	NO	LT GC
8	1437779	45 Y	-	-	-	-	-	NAD	E1V2M5=8	YES	LT FT
9	374559	87 Y	HTN CAD	-	-	-	-	NAD	E2V2M2=6	YES	RT GC
10	1439363	52 Y	HTN	YES	-	-	-	NAD	E1V1M5=7	YES	LT GC
11	1466777	35 Y	-	-	-	-	-	NAD	E1VetM2=3	NO	RT GC
12	1453248	44 Y	HTN	-	-	-	-	NAD	E3V5M6=14	NO	RT GC
13	1439834	50 Y	HTN	-	-	-	-	NAD	E3V2M5=10	YES	LT GC
14	1442840	67 Y	-	-	-	-	-	NAD	E1V2M3=6	NO	LT GC
15	1457358	54 Y	HTN	YES	YES	-	-	NAD	E1V2M4=7	NO	LT THALAMIC
16	1449197	61 Y	HTN	-	-	-	-	NAD	E1VetM2=3	NO	LT GC
17	1442840	67 Y	-	-	-	-	-	NAD	E1V2M5=8	NO	LT GC
18	1471955	53 Y	DM, HTN	YES	-	-	-	NAD	E3V4M6=13	NO	RT GC
19	1475987	42 Y	HYPOTHYROIDISM	-	-	-	-	NAD	E3V1M6=10	NO	LT GC
20	1487818	45 Y	HTN	YES	-	-	YES	NAD	E1VetM3=4	YES	LT THALAMIC
21	1464687	61 Y	HTN	-	-	-	-	NAD	E2V2M5=9	NO	RT THALAMIC
22	1463333	60 Y	HTN	YES	-	-	-	NAD	E4V2M4=10	NO	RT GC
23	1459564	38 Y	CAD	YES	-	-	-	NAD	E3V2M5=10	NO	RT GC
24	1461871	60Y	HTN	YES	-	-	-	NAD	E1VetM2=3	YES	RT GC
25	1476110	65 Y	HTN	-	-	-	-	NAD	E1VetM2=3	NO	LT THALAMIC
26	1488696	41 Y	HTN	YES	-	-	-	NAD	E1VetM2=3	NO	LT GC
27	1491189	65 Y	HTN	-	-	-	-	NAD	E1VetM3=4	YES	LT GC
28	778714	52 Y	DM, HTN	YES	-	-	-	NAD	E2VetM4=6	NO	RT GC
29	1465108	65 Y	HTN, CAD	-	-	-	-	NAD	E2VetM5=7	NO	RT GC
30	1502106	55 Y	-	-	-	-	-	NAD	E4V1M5=10	NO	LT GC
31	1505594	60 Y	HTN	YES	-	-	YES	NAD	E1V1M4=6	NO	LT THALAMIC
32	1502106	55 Y	-	-	-	-	-	NAD	E4V1M5=10	NO	LT GC
33	1553678	83Y	CKD	-	-	-	-	NAD	E1VetM2=3	NO	RT GC
34	1546909	58 Y	-	YES	-	-	-	NAD	E2VetM3=5	NO	LT GC
35	1552656	35 Y	HTN	YES	-	-	-	NAD	E1VetM2=3	NO	RT GC
36	1572990	40 Y	HTN	YES	-	-	-	NAD	E1VetM3=4	NO	RT THALAMIC
37	1558327	44 Y	HTN	-	-	-	-	NAD	E3V3M6=11	NO	LT TP
38	1560440	75 Y	DM, HTN	YES	-	-	-	NAD	E2V2M5=9	NO	RT THALAMIC
39	1537330	30 Y	HTN	YES	-	-	YES	NAD	E4V3M5=12	NO	LT THALAMIC

SR NO	**MRD NO**	**AGE**					**SURGICAL MANAGEMENT**		**ON DISCHARGE**			**FOLLOW UP (AFTER 2 MONTHS)**			
			VOLUME	MS	IVH	HCP	PROCEDURE	DURATION OF SURGERY	GCS	GOS	mRS	GCS	GOS	mRS	
1	1539938	34 Y	51.28	13.5MM	YES	YES	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM3 =5	2	5	E4VtrM4=8	3	5	
2	1540539	79 Y	69.3	5.1MM	YES	YES	EVD INSERTION	30 MINS	E1VtrM2=3	2	5	EXPIRED	-	-	
3	1521287	40 Y	110	10MM	YES	NO	DC WITH HEMATOMA EVACUATION	2.5 HRS	E4VtrM5=9	3	5	E4VtrM6=10	3	5	
4	1531645	40 y	20	6.2MM	YES	YES	EVD INSERTION	30 MINS	E4V4M5=13	3	4	E4V5M6=15	4	2	
5	1527435	65 Y	130.8	12.8MM	YES	YES	EVD INSERTION WITH ICH EVACUATION	3.5HRS	E4VtrM3=7	3	4	E4VtrM5=9	3	3	
6	1511179	45 Y	6.8	7.2MM	YES	YES	EVD INSERTION	45 MINS	E4V2M5=11	3	4	E4V3M6=13	4	2	
7	1442840	67 Y	47	6.8MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4VtrM5=9	3	5	E4V2M6=12	3	4	
8	1437779	45 Y	24.48	12MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4V3M4=11	3	4	E4V3M6=13	3	4	
9	374559	87 Y	68.6	6.7MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	EXPIRED	-	-	-	-	-	
10	1439363	52 Y	69.3	8.6MM	NO	NO	DC WITH HEMATOMA EVACUATION	3.5HRS	E4V3M5=12	3	4	E4V4M6=14	3	4	
11	1466777	35 Y	25.29	6.1MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4VtrM4=8	3	4	E4V2M5=11	4	4	
12	1453248	44 Y	49.5	5.4MM	NO	NO	DC WITH HEMATOMA EVACUATION	2.5HRS	E4VtrM5=9	3	4	E4VtrM5=9	3	4	
13	1439834	50 Y	44	10.2MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4V3M5=12	3	4	E4V4M6=14	4	3	
14	1442840	67 Y	76.6	6.8MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4VtrM6=10	3	4	E4V2M6=12	3	3	
15	1457358	54 Y	32.6	9.3MM	YES	YES	EVD INSERTION	30 MINS	E4V1M6=11	3	4	E4V2M6=12	3	4	
16	1449197	61 Y	91.2	11.3MM	YES	NO	DC WITH HEMATOMA EVACUATION	3.5 HRS	E4V2M5=11	3	4	E4V2M6=12	3	4	
17	1442840	67 Y	76.6	6.8MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4VtrM5=9	3	4	E4VtrM6=10	3	4	
18	1471955	53 Y	12	3.3MM	YES	YES	EVD INSERTION	45 MINS	E4V5M6=15	3	4	E4V5M6=15	4	2	
19	1475987	42 Y	43.61	3.9MM	NO	NO	CRANIOTOMY AND HEMATOMA EVACUATION	2HRS	E4V4M6=14	3	4	E4V5M6=15	3	4	
20	1487818	45 Y	22.44	9.7MM	YES	YES	EVD INSERTION	45 MINS	E4V1M5=10	3	4	E4V2M6=12	3	3	
21	1464687	61 Y	10	2.2MM	NO	YES	EVD INSERTION	30 MINS	E4V5M6=15	3	4	E4V5M6=15	3	3	
22	1463333	60 Y	62.3	10.1MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4V5M6=15	3	4	E4V5M6=15	4	3	
23	1459564	38 Y	21.42	7.6MM	NO	NO	DC WITH HEMATOMA EVACUATION	2.5 HRS	E4V5M6=15	3	4	E4V5M6=15	3	3	
24	1461871	60Y	75.24	7.9MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4VtrM4=8	3	4	E4VtrM5=9	3	4	
25	1476110	65 Y	24	9.8MM	YES	YES	EVD INSERTION	45 MINS	E4VtrM3=7	3	4	E4VtrM5=9	3	4	
26	1488696	41 Y	48.84	11.4MM	YES	YES	EVD INSERTION WITH ICH EVACUATION	3HRS	E4V1M6=11	3	4	E4V4M6=14	4	4	
27	1491189	65 Y	70	6.9MM	YES	YES	CRANIOTOMY AND HEMATOMA EVACUATION	2HRS	E4VtrM5=9	3	4	E4V1M5=10	3	4	
28	778714	52 Y	31	7.7MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E3V4M5=12	3	4	E4V5M6=15	3	4	
29	1465108	65 Y	34	8.3MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4VtrM6=10	3	4	E4VtrM6=10	3	4	
30	1502106	55 Y	29	8.8MM	YES	NO	DC WITH HEMATOMA EVACUATION	3.5HRS	E4V3M6=13	3	4	E4V4M6=14	3	4	
31	1505594	60 Y	27.3	6.1MM	YES	YES	EVD INSERTION	30 MINS	E4VtrM5=9	3	4	E4V2M5=11	3	4	
32	1502106	55 Y	26	8.8MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E4V2M5=11	3	2	E4V3M6=13	3	2	
33	1553678	83Y	41	15MM	YES	YES	EVD INSERTION WITH ICH EVACUATION	3.5HRS	EXPIRED	-	-	-	-	-	
34	1546909	58 Y	31	9.5MM	YES	NO	DC WITH HEMATOMA EVACUATION	2.5HRS	E4V4M6=14	4	2	E4V5M6=15	4	2	
35	1552656	35 Y	40	17.1MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM3=5	2	5	E2VtrM4=6	2	5	
36	1572990	40 Y	20	3.2MM	YES	YES	EVD INSERTION	30MINS	E4V4M6=14	4	3	E4V5M6=15	5	2	
37	1558327	44 Y	44	8.2MM	NO	NO	DC WITH HEMATOMA EVACUATION	2HRS	E4V4M6=14	3	4	E4V5M6=15	4	3	
38	1560440	75 Y	33	7.8MM	YES	YES	EVD INSERTION	30 MINS	E4V4M6=14	3	4	E4V5M6=15	4	3	
39	1537330	30 Y	29	7.2MM	YES	YES	EVD INSERTION	30 MINS	E4V5M6=15	3	4	E4V5M6=15	4	3	

SR NO	**MRD NO**	**AGE**	**SEX**	**OCCUPATION**	**DIAGNOSIS**	**DOA**	**DOS**	**PRESENTATION**						
								HEADACHE	VOMITTING	SEIZURES	FOCAL DEFICIT	LOC	ALTERED SENSORIUM	OTHERS
40	1512452	45 Y	F	HOUSEWIFE	LT GC BLEED	21/05/23	21/05/23	-	YES	-	RT HEMIPARESIS	-	YES	-
41	1514522	58 Y	M	FARMER	RT THALAMIC BLEED	26/05/23	26/05/23	YES	-	-	LT HEMIPARESIS	-	YES	-
42	1524452	66 Y	F	HOUSEWIFE	RT GC BLEED	12/05/23	12/05/23	-	YES	-	LT HEMIPARESIS	-	YES	-
43	1578565	42 Y	M	FARMER	LT THALAMIC BLEED	21/08/23	21/08/23	YES	-	-	-	YES	-	-
44	1586654	51 Y	M	FARMER	RT GC BLEED	06/09/23	06/09/23	-	YES	-	LT HEMIPARESIS	-	YES	-
45	1586523	48 Y	F	HOUSEWIFE	RT THALAMIC BLEED	15/09/23	15/09/23	YES	-	-	-	-	YES	-
46	1596354	61 Y	M	PVT JOB	LT GC BLEED	26/09/23	26/09/23	-	-	-	RT HEMIPARESIS	YES	YES	APHASIA
47	1625241	45 Y	F	HOUSEWIFE	RT GC BLEED	11/10/23	11/10/23	-	YES	-	LT HEMIPARESIS	-	YES	-
48	1632545	52 Y	M	FARMER	LT PAREITAL ICH	18/10/23	18/10/23	-	YES	-	RT HEMIPARESIS	-	YES	-
49	1645256	45 Y	F	GOVT SERVICE	LT THALAMIC BLEED	08/11/23	08/11/23	YES	-	-	RT HEMIPARESIS	-	-	APHASIA
50	1652452	52 Y	M	PVT JOB	RT GC BLEED	22/11/23	22/11/23	-	YES	-	LT HEMIPARESIS	-	YES	-

SR NO	**MRD NO**	**AGE**	**COMORBIDITY**	**ADDICTION**				**SYSTEMIC EXAMINATION**	**NEUROLOGICAL EXAMINATION**		**INVESTIGATION (RADIOLOGICAL PARAMETERS**
			-	ALCOHOL	SMOKING	NSAID INTAKE	DRUG ABUSE	-	GCS	PUPILLARY ASYMMETRY	LOCATION
40	1512452	45 Y	HTN	-	-	-	-	NAD	E1VetM3=4	YES	LT GC
41	1514522	58 Y	DM, HTN	YES	-	-	-	NAD	E1V2M5=8	NO	RT THALAMIC
42	1524452	66 Y	HTN	-	-	-	-	NAD	E1V2M3=6	NO	RT GC
43	1578565	42 Y	HTN	YES	-	-	-	NAD	E2V3M5=10	NO	LT THALAMIC
44	1586654	51 Y	DM, HTN, CKD	YES	-	-	YES	NAD	E1VetM4=5	YES	RT GC
45	1586523	48 Y	HTN	-	-	-	-	NAD	E2V2M4=8	NO	RT THALAMIC
46	1596354	61 Y	HTN	YES	-	-	-	NAD	E1V1M5=7	NO	LT GC
47	1625241	45 Y	DM, HTN	-	-	-	-	NAD	E1V2M5=8	YES	RT GC
48	1632545	52 Y	HTN	-	-	-	-	NAD	E2V3M5=10	NO	LT PARIETAL
49	1645256	45 Y	HTN	-	-	-	-	NAD	E2V4M5=11	NO	LT THALAMIC
50	1652452	52 Y	DM, HTN, CKD	YES	-	-	YES	NAD	E1VetM3=4	YES	RT GC

SR NO	**MRD NO**	**AGE**					**SURGICAL MANAGEMENT**		**ON DISCHARGE**			**FOLLOW UP (AFTER 2 MONTHS)**			
			VOLUME	MS	IVH	HCP	PROCEDURE	DURATION OF SURGERY	GCS	GOS	mRS	GCS	GOS	mRS	
40	1512452	45 Y	60	10MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E3V1M5=9	3	4	E4V2M5=11	3	4	
41	1514522	58 Y	26.6	6.6MM	YES	YES	EVD INSERTION	45 MINS	E4V3M5=12	3	4	E4V5M6=15	3	4	
42	1524452	66 Y	44.2	9.2MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM4=6	2	5	E4VtrM4=8	3	4	
43	1578565	42 Y	29.5	6.6MM	YES	YES	EVD INSERTION	30 MINS	E4V4M6=14	3	4	E4V5M6=15	4	3	
44	1586654	51 Y	62.5	11.1MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM5=7	3	4	E4VtrM5=9	3	4	
45	1586523	48 Y	28.5	6.6MM	YES	YES	EVD INSERTION	30 MINS	E4V4M6=14	3	4	E4V5M6=15	4	3	
46	1596354	61 Y	55.5	9.8MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM4=6	2	5	E3VtrM5=8	3	5	
47	1625241	45 Y	48.5	10.2MM	YES	NO	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM5=7	3	4	E4VtrM5=9	3	4	
48	1632545	52 Y	36.3	6.8MM	NO	NO	CRANIOTOMY AND HEMATOMA EVACUATION	2 HRS	E3V3M5=11	3	4	E4V5M6=15	3	4	
49	1645256	45 Y	26.5	7.6MM	YES	YES	EVD INSERTION	45 MINS	E4V4M6=14	3	4	E4V5M6=15	4	3	
50	1652452	52 Y	54.5	11.4MM	NO	NO	DC WITH HEMATOMA EVACUATION	3HRS	E2VtrM5=7	2	5	E4VtrM5=9	3	4	

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The Principal
Dayanand Medical College & Hospital
Ludhiana

Subject:: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Jadhao Atul Rakhmaji**, a student of your college for **MCh Neuro-Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
To study the surgical outcomes in patients with hypertensive supratentorial intracerebral hemorrhage .

Supervisor	**Co-Supervisor:-**	**Co-Supervisor:-**
Dr. Shivender Sobti	Dr. Hanish Bansal	Dr. Birinder Singh Paul
Prof.,	Assoc. Professor	Professor
Dept. of Neuro-Surgery	Dept. of Neuro-Surgery	Dept. of Neurology

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED
IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE DEGREE OF

M.Ch.
In
NEUROSURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

OCTOBER, 2022

DR. JADHAO ATUL RAKHMAJI

DEPARTMENT OF NEUROSURGERY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE
For the degree of	M.Ch. Neurosurgery
Name of the candidate	Dr. Jadhao Atul Rakhmaji
Supervisor	Dr. Shivender Sobti, Professor, Department of Neurosurgery, DMCH, Ludhiana
Co-supervisor(s)	Dr. Hanish Bansal, Associate Professor, Department of Neurosurgery, DMCH, Ludhiana Dr. Birinder Singh Paul, Professor, Department of Neurology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

INTRODUCTION: Intracerebral Haemorrhage is a serious disease with high mortality and morbidity. Intracerebral haemorrhage (ICH) ranks second among pathologies underlying stroke. The Incidence is 108-172 per year per lakh population.

AIMS & OBJECTIVES: To Study the Outcomes of Surgical Interventions in Patients with Hypertensive Supratentorial Intracerebral Haemorrhage.

MATERIAL & METHODS: The study will be conducted on patients who will be diagnosed as a case of Hypertensive Supratentorial Intracerebral Haemorrhage in Dayanand Medical College and Hospital from August 2022 to July 2023. The patients will have to satisfy the inclusion criteria and none of the exclusion criteria. The data collected will be compiled and analysed to study the Outcomes of Surgical Management hypertensive Supratentorial intracerebral haemorrhage. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21 version (SPSS Inc., Chicago IL, USA) statistical program for Microsoft windows.

CLINICAL SIGNIFICANCE: The knowledge the Outcomes of Surgical Management hypertensive Supratentorial intracerebral haemorrhage will help in better management of the patients having hypertensive intracerebral haemorrhage and thus improving the outcome in these patients.

KEYWORDS: INTRACEREBRAL HAEMORRHAG (ICH), SUPRATENTORIAL.

(Signature) Candidate	(Signature) Supervisor	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE"** exist in the Department of Neurosurgery, Dayanand Medical College, Ludhiana & will be provided to the candidate. We will see that the data being included in the thesis are genuine & is collected by the candidate himself under our supervision & guidance.

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Place: LUDHIANA

Date: 22/11/2022

APPROVAL PROFORMA BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL,
LUDHIANA

Name of candidate	Dr. Jadhao Atul Rakhmaji
Department	Neurosurgery
Topic of Thesis	TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE
Likely date of appearing in PG examination	May/June 2025
Date of enrollment	13th April 2022
Name of Head of Department	Dr. Ashwani K Chaudhary
1) Supervisor **2) Co-Supervisor(s)**	Dr. Shivender Sobti Dr. Hanish Bansal Dr. Birinder Singh Paul
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / No

Prof. Arvind Malhotra
Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR. JADHAO ATUL RAKHMAJI R D JADHAO, NEAR MATRUBHUMI GYM, RAM NAGAR BULDHANA- 443001 MAHARASHTRA.
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA
3	COURSE OF STUDY AND SUBJECT	M.CH. NEUROSURGERY
4	DATE OF ADMISSION OF COURSE	13TH APRIL 2022
5	TITLE OF THE TOPIC	TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE

6.0 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR STUDY

Intracerebral Haemorrhage is a serious disease with high mortality and morbidity. Intracerebral haemorrhage (ICH) ranks second among pathologies underlying stroke.[1] The Incidence is 108-172 per year per lakh population.[2]

ICH constitutes about 15 % of the total number of strokes. A considerable number of ICH is misdiagnosed as brain infracts[3] resulting in an underestimation of the incidence of ICH in any community. Now more than half of the acute stroke patients are investigated within 3 days resulting in identification of more hemorrhagic strokes.

Arterial hypertension is reported as the main cause of ICH – 57% in metanalysis.[4] The frequency varies considerably. Neurosurgical case series show more hematomas from aneurysms and arteriovenous malformations (AVM) than from hypertension.[5] More anticoagulant bleeds will be observed where anticoagulant therapy is widely used. Charcot and Bouchard (1868)[6] described miliary aneurysms, which is the pathological substrate of many of these haemorrhages. The underlying pathology is lipohyalinosis and fibrinoid necrosis.[7]

The outcome of this common and distinct entity of Hypertensive ICH is determined mainly by severity of bleeding.[8] Previous multivariate studies principally investigated short term outcome – 30-day mortality[9] after ICH and showed that age, level of consciousness, volume of hematoma and presence of intraventricular blood are independent predictors of recovery.[10] It is not clear whether alcohol consumption, smoking, use of NSAIDs or clinical parameters like level of blood pressure, target organ damages like hypertensive changes in optic fundus and left ventricular hypertrophy would influence the outcome. These factors might also impair the outcome by making patients prone to severe initial bleeding, to enlargement by hematoma or to cerebral ischemia. Further, prognostic significance of signs of tentorial herniation, midline shift and interference

of the hematoma with the CSF circulation has to be evaluated. The impact of surgical evacuation of hematoma on the prognosis, selection criteria and timing of evacuation has to be assessed.[11]

Modifiable risk factors that increase the incidence of stroke include hypertension, smoking, alcohol, diabetes, dyslipidemia beside some non-modifiable risk factors.[12] Attempts are being made to device optimal management strategies and to identify those who stand a poor prognosis with the available investigation and treatment options.

Increasing age, low level of consciousness, low GCS score, abnormalities in arterial blood pressures, temperature, respiration at presentation, severity of weakness, speech disturbances, gaze palsies, brainstem involvement, higher volume of ICH, infratentorial origin of ICH, intra-ventricular or sub-arachnoid extension of ICH, midline shift, higher ICH score have all been studied by various workers as important markers of bad prognosis after ICH.[13] Beside these, the presence of one or more co-morbid condition as well as some biochemical parameters at presentation has been found to influence outcome.

With the arrival of computed tomography of the brain, diagnosis of intra-cerebral haemorrhage has been taken from the age of calculated speculation with details of clinical features, angiograms and ‘bloody taps’ to the present day of arrival of a definitive diagnosis in a matter of minutes. A significant reason for the decreasing trend of mortality in the intra-cerebral haemorrhage patients in the industrialized countries have also been due to the identification of the factors which might adversely affect the outcome, stratifying patients and instituting prompt acute stroke care.

6.2 REVIEW OF LITERATURE-

Hypertensive ICH is a distinct clinical entity with a prevalence of 10-30 per lakh population.[14] Primary intracerebral haemorrhage occurs more frequently in men than women,[15] particularly those older than 55 years of age,[16] and in certain populations as blacks and Japanese.[16] Hypertension is the single most important risk factor in middle aged and elderly person.[17-18] The increased risk of intracerebral haemorrhage is substantially greater among those who have ceased their antihypertensive medication, are relatively young or are current smokers.[18] This is the mundane but often devastating "spontaneous" brain haemorrhage. More cases are diagnosed now with increasing use of CT and MRI scans. With improved antihypertensive treatment, the peak age of occurrence is increasing from 60-70 years to 71-75 year. Spontaneous intracranial haemorrhage comprises 10-20% of stroke occurrences.[19] Intracerebral haemorrhage is associated with higher mortality rate with only 38% of affected persons surviving in the first year.[20] Ruptured saccular aneurysm and vascular malformation, and haemorrhage associated with the use of anticoagulants or thrombolytic agents, cerebrovascular amyloidosis and acquired or congenital bleeding disorders are the other causes of intracerebral haemorrhage.[21,22] Nevertheless, the hypertensive intracerebral haemorrhage serves as a model for understanding and managing other cerebral haemorrhages.

Though medical management of intra-cerebral haemorrhage has been the way, surgical management has been known since the times of McEwen, who performed the first successful operation for intra-cerebral haematoma in 1883. Though no definite guidelines exist to decide between the two lines of management, the increasing availability of minimally invasive techniques like stereo-tactic aspiration may make this modality indispensable in the days to come.

But all is not well for a country like India, where there still exist pockets where CT brain is unheard of, stroke care centers unimaginable, and

supply almost always short of demand. Thereby, identifying definite risk factors, designing and implementing policies to contain them, making prompt and accurate diagnosis, stratifying patients according to outcome predictors, and thus ensuring prompt referral of deserving critical patients to tertiary centers for intensive management, may be the need of the hour as we wait to embrace the newer advances into our management protocols. Intracerebral hemorrhage accounts for approximately 10% of strokes and its clinical importance derives from its frequency and accompanying high mortality.

Although the latter is strongly dependent on HEMATOMA SIZE and to a lesser extent, LOCATION, the overall mortality rate varies between 25% and 60%.[23] There has been a general decline since the 1980s in the incidence of stroke, including ICH, as a result of improved detection and treatment of hypertension. However, ICH continues to be a major health problem especially in those who lack hypertension treatment and the genetically predisposed.

A growing body of evidence suggests that genetic factors, such as the possession of the E2 and E4 alleles of the apolipoprotein E, play an important role in the occurrence of certain forms of ICH, such as lobar hemorrhages (O'Donnell et al., 2000).[24] Novel potential genetic factors predisposing to ICH continue to be added by experimental and clinical studies (Gould et al., 2006).[25]

Although it would appear intuitively that evacuation of a hematoma might be beneficial, surgical results have not been superior to those with medical measures alone (Waga and Yamamoto; Batjer et al; Juvela et al; Rabinstein et al).

A large, multicenter, randomized study involving 1,033 patients with supratentorial hemorrhage, under the auspices of the Surgical Trial in Intracerebral Haemorrhage (STICH) study reported by Mendelow and colleagues, has failed to show a benefit from early surgery on survival or neurologic functioning at 6 months. This negative result extended to

almost all levels of neurologic deficit and all age groups. In a post hoc analysis, clots that were small and close to the surface of the brain may have benefited from evacuation.

As a result, this approach has been virtually abandoned, but in a few instances with ongoing deterioration in young patients with hematomas that are easily accessible from the cortical surface, evacuation of the clot has been undertaken.[26] In contrast, the surgical evacuation of cerebellar hematomas is a generally accepted treatment and is a more urgent matter because of the proximity of the mass to the brainstem and the risk of abrupt progression to coma and respiratory failure.

Mayer and coworkers studied the promising approach of administering clotting factor VII within 4 h of hemorrhage. In a preliminary study (phase I&II), survival was improved and there was a reduction in enlargement of the hematoma, but their subsequent series (phase III FAST trial) has failed to confirm the benefit on survival so that infusion of factor VII is not currently part of routine practice.[27] Therefore, the surgical management of ICH is debatable because the assessment of potential interventions awaits the completion of properly designed prospective clinical trials.

6.3 AIMS AND OBJECTIVES-

To Study the Outcomes of Surgical Interventions in Patients with Hypertensive Supratentorial Intracerebral Haemorrhage.

7.0 MATERIAL AND METHODS:

7.1 SOURCE OF DATA:

The patients presenting to Dayanand Medical College and Hospital, Ludhiana, with Hypertensive Supratentorial Intracerebral Haemorrhage and got operated will be taken into study group. Study will be conducted from 1st December 2022 to 30th November, 2023.

7.2 METHOD OF COLLECTION OF DATA

- All the patients more than 18 years will be evaluated neurologically and radiologically with non-contrast CT of head.
- Detailed clinical and neurological examinations will be performed in all patients
- All relevant information like Demographic (Age, Sex, Address), Family history, Personal History Co-morbidity etc. will be collected

INCLUSION CRITERIA-

- All patients above 18 years of age with Supratentorial Hypertensive intracerebral haemorrhage who will require surgical intervention.

EXCLUSION CRITERIA-

- Head trauma
- Intracranial neoplasm
- Aneurysm
- AVM
- Bleeding diathesis
- Infratentorial haemorrhage
- Not willing for surgery

OUTCOME:- Outcome will be assessed by using GOS (Glasgow outcome scale),GCS (Glasgow coma scale) at the time of discharge and on follow up at 2 months and mRS (Modified Rankin scale) at the time of discharge and on follow up at 3 months.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

Management plan

Patients will be managed according to the clinical and radiological findings and appropriate Surgical procedure will be choosen (Craniotomy and hematoma evacuation, Decompressive Craniectomy and hematoma evacuation, EVD insertion with ICH evacuation, EVD insertion). ICU care and ventilator and ionotropic support will be given as required.

Indications for Surgery:

1. Patients with Supratentorial ICH of size ≥ 2 cm of diameter and with GCS of ≥ 5.[29]
2. Spontaneous Lobar ICH on CT scan (≤ 1cm from the cortical surface of the brain)[30]
3. Temporal Hematoma as small as 30ml[31]
4. Hematoma occupying 4-8% of intracranial space with clinical status of the patient.[31]
5. Hematoma occupying 8-12% of intracranial space[31]
6. Hematoma volume of 50-100ml with evidence of raised ICP (Pupillary asymetry, and drop in GCS)

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO

7.4 HAS ETHICAL CLEARENCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

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PROFORMA

Sr No: -

Name: Age: Sex:

Address: Occupation:

MRD No: DOA:

Diagnosis: DOS: Procedure:

PARAMETERS ON ADMISSION

Clinical parameters

1. Presenting symptom:

Headache:

Vomiting:

Seizures:

Focal deficit:

Altered sensorium:

Others:

2. Systemic examination

3. Neurological examination

- GCS:
- Pupillary asymmetry:
- Hemiparesis

4. Presence of co-morbidity:

5. History of Alcoholism:

Smoking:

NSAID intake:

Drug abuse:

6. Radiological parameters:

Location of hemorrhage:

Volume of hematoma: _____ ml

Midline shift: _____mm

Associated IVH:

Hydrocephalus:

Percentage Of hematoma occupying intracranial space:

HOSPITAL COURSE:

Surgical Management:

1. Craniotomy and Hematoma evacuation
2. Decompressive craniectomy with Hematoma evacuation
3. EVD insertion with ICH evacuation
4. EVD insertion

- Duration of Surgery
- Anesthesia Given

PARAMETERS ON DISCHARGE

GCS (Glasgow coma scale):

GOS (Glasgow outcome score):

mRS (Modified rankin score):

FOLLOW UP (after 2 months)

GCS (Glasgow coma scale):

GOS (Glasgow outcome score):

mRS (Modified rankin score):

10. Any other:

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I…… D/o, W/o, S/o……. am willing to participate in the study **"TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out of the study any time without assigning any reason.

…….. ……

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीजअनुमतिफार्म

तिथि_____

मैं_____ पत्नी/पुत्र/पुत्री_____

निवासी_____ इस शोध कार्य **"TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE"** में भाग लेने के लिए सहमत हूँ। मैं इस शोध कार्य में भाग लेने के लिए अपनी स्वेच्छासे तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और नही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोध कार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृतिव्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृतिव्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ_____

“TO STUDY THE SURGICAL OUTCOMES IN PATIENTS WITH HYPERTENSIVE SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾਹੀ ਕੋਈ ਦਬਾਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾਮਨੇਰਥ ਸਮਝਾਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈਸ ਕਦਾਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾ ਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ_____ ਪਤਾ_____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ